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ABSTRACT

These proceedings from the second conference on the Epidemiology of Aging sponsored by the National Institutes of Health indicate the direction of recent research and concern in this area. Attempts are made to formulate a definition of aging other than that of chronological age. A number of biomedical correlates of aging are addressed, including some predictors of longevity, as well as age-correlated diseases that constitute some major health problems for older people. The social and psychological correlates of aging are examined as two highly productive research areas in the field. Health care implications and problems of the aged are addressed according to demographic projections and morbidity estimates. A summary of recent advances is provided, and major research concerns regarding the problems of the aged are identified. (Author/[illegible])

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Second Conference on the Epidemiology of Aging

U.S. DEPARTMENT OF HEALTH,
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NATIONAL INSTITUTE OF
EDUCATION

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Second Conference
March 28-29,1977
National Institutes of Health
Bethesda, Md.

Editors
Suzanne G. Haynes, Ph.D.
Manning Feinleib, M.D., Dr. P.H.

Associate Editors
Jane A. Ross, [illegible]
Lorraine Stallones, M.P.H.

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The front cover represents
the growth of the older pop-
ulation in the U.S. The actual
data may be seen in figure II-4
of the conference proceedings.

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Washington, D.C. 20402

Participants

Richard C. Adelman, Ph. D.
Temple University Institute on Aging
[illegible] N. Broad Street
Philadelphia, PA [illegible]

Reubin Andres, M.D.
Gerontology Research Center
National Institute on Aging
Baltimore City Hospitals
Baltimore, MD 21224

Robert C. Atchley, Ph. D.
Scripps Foundation Gerontology Center
Miami University
Oxford, OH 45056

Lawrence G. Branch, Ph. D.
Department of Preventive and
Social Medicine
Harvard Medical School
25 Shattuck Street
Boston, MA 02115

[illegible] F. [illegible], Ph. D.
Social Security Administration
Meadows East
6401 Security Blvd.
Baltimore, MD 21235

Jacob A. Brody, M.D.
National Institute on Aging
National Institutes of Health
Bethesda, MD 20205

Robert N. Butler, M.D.
National Institute on Aging
National Institutes of Health
Bethesda, MD 20205

Judith Cohen, Ph. D.
University of California
School of Public Health
Berkeley, CA 94720

Paul T. Costa, Jr., Ph. D.
Gerontology Research Center
National Institute on Aging
Baltimore City Hospitals
Baltimore, MD 21224

Manning Feinleib, M.D., Dr.P.H.
National Heart, Lung, and
Blood Institute
National Institutes of Health
Bethesda, MD 20205

Joseph F. Fraumeni, Jr., M.D.
National Cancer Institute
National Institutes of Health
Bethesda, MD 20205

Don C. Gibson, D.V.M., M.P.H.
National Institute on Aging
National Institutes of Health
Bethesda, MD 20205

Robert S. Gordon, M.D.
National Institutes of Health
Room 118, Building 1
Bethesda, MD 20205

Samuel W. Greenhouse, Ph. D.
Department of Statistics
George Washington University
Washington, D.C. [illegible]

Ernest M. Gruenberg, M.D., Dr. P.H.
Johns Hopkins University
School of Hygiene and Public Health
615 North Wolfe Street
Baltimore, MD 21205

[illegible] [illegible], M.D.
Tokyo Metropolitan Institute
of Gerontology
35-2 Sakaecho, Itabashi
Tokyo, Japan 173

Suzanne G. Haynes, Ph. D.
National Heart, Lung, and
Blood Institute
National Institutes of Health
Bethesda, MD 20205

[illegible] [illegible], [illegible], Ph. D.
Department of [illegible]
Duke University Medical
Center
Durham, NC 27710

[illegible] [illegible], M.D.
[illegible] Street, N.W.
Washington, D.C. [illegible]

[illegible] [illegible], M.D.
Office of the Surgeon
General
Department of the Army
The Pentagon, Room [illegible]
Washington, D.C. 20310

William B. Kannel, M.D., M.P.H.
Boston University
School of Medicine
80 Concord Street
Boston, MA 02118

Stanislav V. Kasl, Ph.D.
Yale University
School of Medicine
60 College Street
New Haven, CT 06510

Mary Grace Kovar
National Center for Health
Statistics
3700 East-West Highway
Hyattsville, MD 20782

Florence V. Mahoney
3600 Prospect Street, N.W.
Washington, D.C. 20007

Takashi Makinodan, M.D.
Department of Medicine
University of California
Los Angeles, CA 90024

Bernard Nash
National Retired Teachers
Association and American
Association of Retired Persons
[illegible] Connecticut Avenue
Washington, D.C. [illegible]

Adrian M. Ostfeld, M.D.
Yale University
School of Medicine
60 College Street
New Haven, CT 06510

Erdman Palmore, Ph.D.
Center for the Study of Aging
and Human Development
Duke University Medical Center
Durham, NC 27710

[illegible], M.D.
National Institute of Child
Health and Human Development
National Institutes of Health
Bethesda, MD 20205

George Sacher
Argonne National Laboratories
Division of Biological and Medical
Research
Argonne, IL 60439

Edward Schneider, M.D.
Gerontology Research Center
National Institute on Aging
Baltimore City Hospital
Baltimore, MD 21224

Ethel Shanas, Ph.D.
Department of Sociology
University of Illinois
Chicago Circle Campus
Chicago, IL 60680

Jacob S. Siegel
Population Division
U.S. Bureau of the Census
Washington, D.C. 20233

[illegible], M.P.H.
National Heart, Lung, and
Blood Institute
National Institutes of Health
Bethesda, MD 20205

[illegible], Ph.D.
University of Pennsylvania
Leidy Lab/G7
Department of Biology
Philadelphia, PA 19104

Marc E. Weksler, M.D.
Cornell University Medical
College
The New York Hospital
1300 York Avenue
New York, NY 10021

[illegible], M.D.
Department of Psychiatry and
Behavioral Sciences
University of Washington
School of Medicine
Seattle, WA 98195

Preface

A conference on the Epidemiology of Aging was held 5 years ago at Elkridge, Maryland, under the chairmanship of Dr. Adrian Ostfeld. At that time, aging studies were part of the National Institute of Child Health and Human Development. Now that the National Institute on Aging has become a reality, Dr. Robert Butler, Director of the Institute, thought it appropriate to review the state of the art in order to identify the needs in the epidemiology of aging, one of the major programs of the Institute.

An indication of the direction of recent research and concern may be obtained from a brief scanning of the program of this conference. The first session, chaired by Dr. Samuel Greenhouse, constituted an attempt to formulate a definition of aging other than that of chronological age. This problem has bothered epidemiologists and other researchers tremendously. There is no doubt that alternate definitions would be a major step forward.

The next session, chaired by Dr. George Sacher, addressed a number of biomedical correlates of aging, including some of the predictors of longevity. Material was presented from several longitudinal studies, including the ongoing National Heart, Lung, and Blood Institute Framingham study which has, in many respects, developed into an aging study. Consideration was also given to some of the diseases that are known to correlate strongly with age and that constitute some of the major health problems of older people.

Following the discussion on the biological correlates, Dr. Judith Cohen chaired a session on the social and psychological correlates of aging. This area was not adequately covered in any formal sense at the conference 5 years ago. It is probably one of the more productive areas that has appeared in the intervening years.

Dr. Don Gibson, co-chairman of the Elkridge conference, then chaired a session on the health care implications and problems of the aged. These included demographic projections, morbidity estimates, and other aspects of health care problems in this segment of our population.

Finally, there was an attempt to summarize in a broad sense what we have learned from this conference, to review the strides made since the earlier conference, and to identify major gaps in our knowledge of the problems of the aged. Thus, it is hoped that the information presented here will be a basis for the emerging program in epidemiology, biostatistics and demography at the National Institute on Aging.

Editors
Suzanne G. Haynes, Ph.D.
Manning Feinleib, M.D.,
Dr. P.H.

Associate Editors
Jane A. Ross, M.A.
Lorann Stallones, M.P.H.

Technical Assistance
Ida L. Hoffman, M.A.
Ruth R. Dalton

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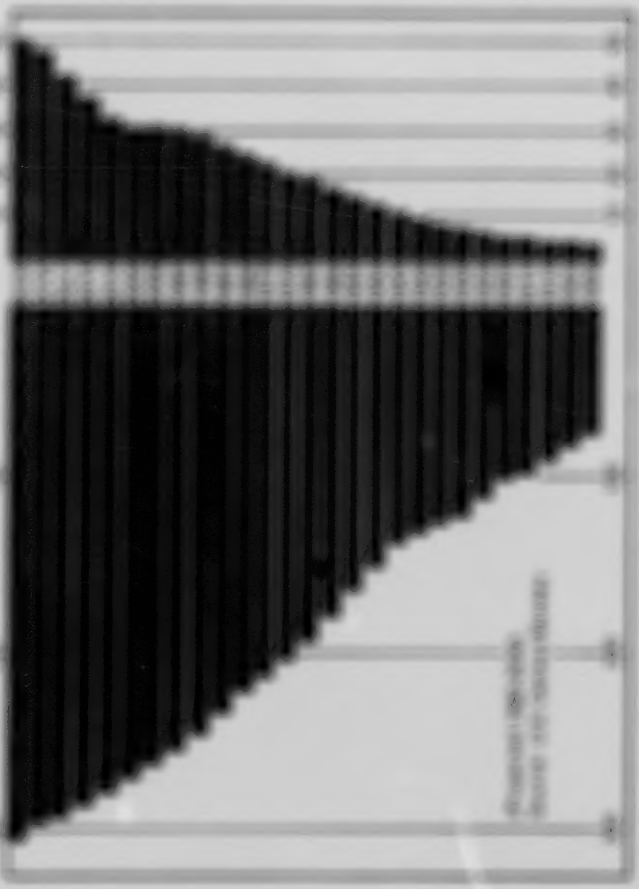

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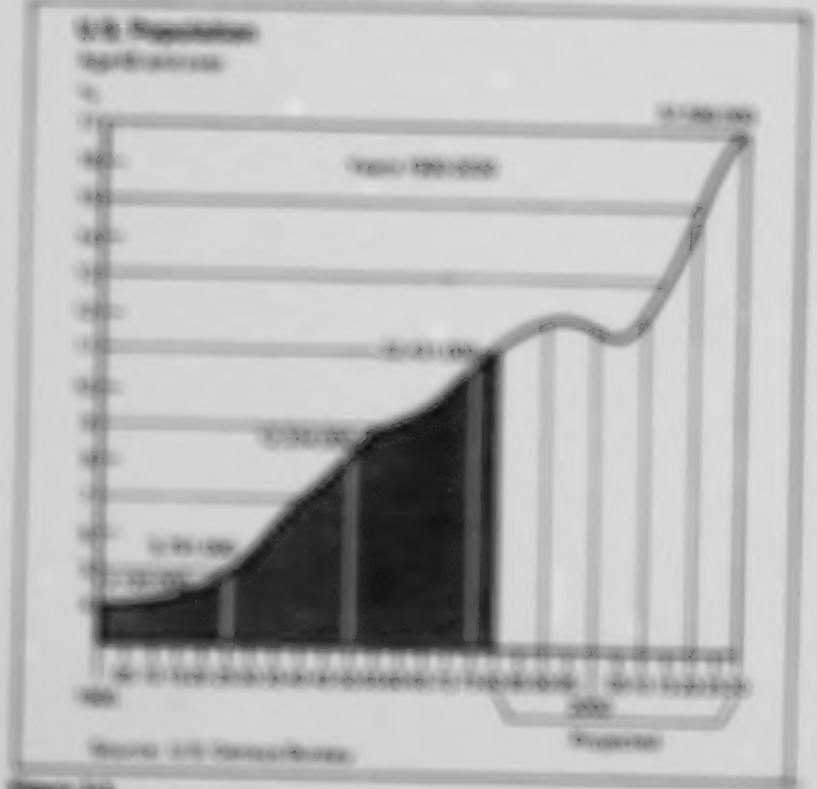

Source: U.S. Census Bureau.

Figure 1.2
U.S. population: Persons age 65 and over, 1900–2050

American women [illegible] tend to marry men an average of 3 years older than themselves, they face approximately 11 years of widowhood.

It would [illegible] to [illegible] a [illegible] to [illegible] the importance of epidemiology to the developing Institute. In light of our particular mission, we felt that we could not necessarily follow the traditional program of organization of the other National Institutes of Health. We are therefore moving toward a central planning group consisting of the director, deputy director, scientific director, associate director of Extramural and Collaborative Research, and associate director of Epidemiology, Demography, and Biometry. This group seems to us to represent a balance between biology and epidemiology, thus making it possible for us to consider the broad character of our population in planning what research is needed to improve the quality of their lives.

At a conference I recently attended in New York on Future Directions in Health Care, no mention was made of the future size of the elderly population to be served until I did so at the end of the meeting. All the planning of future directions of health care did not include any consideration of the character, demographic structure, and disease patterns of the population that we will have 20, 30, or 40 years from now.

We often hear reference made to the "aging problem" and, in many instances, we can observe the tragedy associated with growing old in America. This is particularly the case with older women, who may be impoverished, vulnerable to crime, and afflicted with a variety of diseases such as osteoporosis. We are all aware of the considerable impact of Alzheimer's disease-senile dementia, which afflicts so many older Americans and is a contributing factor in a large number of nursing home admissions in this country.

If we think back for a moment, we see that in this century a major change has taken place in both the absolute number and relative proportion of older people. This change should be regarded as a triumph; the society has always wanted people to have the opportunity to live longer. Unfortunately, our society was unprepared for this "demographic revolution"; we did not have the social institutions, the medical care systems, the employment policies, and the nursing homes to properly respond to the change. This is only a temporary disequilibrium and should not be viewed as fatal. We are already showing signs of adapting by improving the socioeconomic conditions and overcoming many of the prejudices that exist toward older people. This, mainly, of course, as far as the Institute is concerned, we must acquire new knowledge. It is only by conducting research that we will discover new knowledge to advance our understanding of the underlying causes of the aging process and help us separate disease from aging. The improvements in health care and service delivery that can result from the application of this new knowledge will improve the quality of life of the old.

We now have about 1.3 million people in nursing homes in this country, 800,000 of whom are over 80 years of age. Unless we advance our understanding of the diseases which bring people into these nursing homes, we can expect the present number to increase to 3.6 million people in about 40 years. To avoid such a situation, we need to develop an analytic epidemiology which can contribute to a better understanding of the precursors of these conditions and then to mount an effective biomedical, social, and behavioral studies program on aging here at NIH.

Session I

Definitions of Aging

Opening Remarks by Chairperson Samuel W. Greenhouse, Ph. D.

In the Conference on the Epidemiology of Aging, held 5 years ago, a very interesting comment was made by Dr. Adrian Ostfeld who is also attending this conference. He stated, "But the challenge of applying epidemiologic methods to the study of aging is a far more difficult one than applying them to a disease. A clear, valid and reliable definition of aging remains to be formulated."[1] I assume that that statement is a prelude to the first session of this particular meeting which is concerned with the definitions of aging.

May I emphasize that the task of arriving at a definition of the epidemiology of aging is no less difficult. Perhaps "definition" is not the right word. More appropriate would be the formulation of the appropriate questions and objectives that are to be investigated by epidemiologic research. There are probably some who still believe that epidemiologic studies of aging relate to the epidemiology of diseases occurring in the aged. However, I am sure many here will agree with me when I say that the epidemiology of aging should extend far beyond such studies. The problem, though, is not clear—what should the prescribed area of research be? What systems of the human organism should be studied—the immunologic, the circulatory, neurological, behavioral, psychological—probably all—but then what are the priorities? At what age should we begin these studies? We now understand enough to reject age 65. At this age we see the consequences of the aging process. Should it then be age 40 or 30 or even birth? Much current epidemiologic research relates to risk factors. Risk factors for abnormal aging? Then, what is normal aging?

Clearly, these questions are only some of many more than can be posed. We look forward to this conference for answers to these questions and for the groundwork of an important and effective program of research in the epidemiology of aging.

We begin this morning's session by hearing from Dr. Robert Butler, director of the National Institute on Aging, who will present a summary of current definitions of aging.

Current Definitions of Aging

Robert N. Butler, M.D.

National Institute on Aging
National Institutes of Health
Bethesda, MD 20205

This presentation is essentially a commentary on the problems of defining aging. To begin with, there is no denying the reality of aging, a process often described as intrinsic, deleterious, universal, progressive, and irreversible. One creative approach that has proved to be an important research strategy is the separation of the aging process from the diseases and other environmental factors that may accompany it. For example, at the time of our studies of human aging conducted here at NIH in the 1950's, people thought of arteriosclerosis as being part of aging. Today, we know that arteriosclerosis is a disease; in fact, there are those who would even call it a pediatric disease, as one may find plaques in the thoracic aorta of the child.

The problem of defining aging is complicated by variations in how different people age, both physiologically and psychologically, as well as the differences in the aging process in systems, organs, and tissues.

The term "old age" itself is by no means constant. The chronological demarcation using age 65 as the dividing line was begun by Bismarck in the 1880's in legislation dealing with retirement pensions. Today we have entitlements and eligibilities sponsored by our government in which the classification of old age varies. For instance, under Title VII of the Older Americans Act—the nutrition program—60 is the lower limit for the aged; in one HUD housing program (the 202 program), it is 62; and in the Medicare program it is 65.

If we move to the biological definition of aging, which Dr. Adelman will discuss in detail, we can say that aging begins at—in fact, before—conception. The impact of maternal age upon Down's syndrome supports this argument. The Nobelist Medawar, on the other hand, approaches the issue from the perspective of evolutionary theory and would have us consider the entire postreproductive period as constituting aging.

We should look at certain specific systems—immune competence, for example—which merit particular attention. Studies have suggested that people over 60 may have as little as one-tenth the immune competence of those in their teenage years. In addition, we have intellectual competence to consider. Dr. Greenberg will discuss that in connection with the question "Is senile organic brain disease inevitable with age?" Or is there some suggestion through epidemiological studies that after age 80 one may actually have a lower age-specific case rate of Alzheimer's disease and other organic states? Finally, we must consider stress competence, i.e., people's capacity to respond to life's changes.

[illegible], [illegible], [illegible] and others have worked on the epidemiologic aspects of this mortality.

We should view the middle years as a period of transition. Although discussion up to this time has centered on individuals age 65 and above, the Research on Aging Act that created the National Institute on Aging does indicate the appropriateness of our looking at the middle as well as the later years. The middle years are especially important in the study of differences between the sexes and age groups in susceptibility to disease.

This brings us to the problem of risk groups. We do not know enough about the differences in aging associated with individuals' genetic makeup and their early life histories.

To take all these factors into consideration requires a multidisciplinary approach. I think we agree that we cannot look at nature simplistically. Aging is complex, with many antecedent factors leading to specific outcomes of various adaptive levels. We do not have a variety of sophisticated and valid methodological and statistical techniques available to help us weigh the relative contribution of each variable to an outcome. It also seems to me that where the definition of aging is concerned, epidemiology must relate to interactive, prospective and analytic procedures.

If we define aging broadly as a vulnerability or functional susceptibility, we can gain insights into various disease states and their interactions. For example, we must investigate how biological factors contribute to the timing of the onset and the severity of various diseases. I also propose that we must be much more precise than we have been in our use of the term "old" to designate the age group 65 and above. There is a huge amount of territory between ages 65 and 90, a total of 25 years which have not been adequately observed or characterized. We can begin to define aging more precisely by simply specifying narrower age ranges—60 to 64, 65 to 69, 70 to 74, and so on. These categories will be further refined as research on aging continues and our understanding of the aging process increases.

Definition of Biological Aging*

Richard C. Adelman, Ph. D.

Temple University Institute on Aging
3420 N. Broad Street
Philadelphia, PA 19140

It was my responsibility to propose a definition of biological aging which would presumably be of some use in future studies on the epidemiology of aging. The reason I chose to accept this responsibility specifically relates to my somewhat outspoken position that biologists are not yet prepared to make any such definition. Therefore, what I shall do today is pose questions which, at least in my view, are of the most fundamental concern in the biology of aging, outline in a very general sense the types of experimental approaches which probably are necessary in order to evolve answers to these questions, and present brief highlights of one particular experimental approach out of my own laboratory to show both the potential and the pitfalls in pursuing these types of problems.

First of all, with regard to the fundamental question of aging, what is meant by biological aging? Does it mean that specific populations are characterized by finite maximal life spans which can differ enormously from one population to another? Do all members of a given population eventually undergo a progressive decline in their capability for physiological performance as time passes? Do these fundamental features of aging populations relate to one another? Thus far, such issues are totally unresolved.

Whatever the mechanisms responsible for the biology of aging, do they reside within all of the cells that comprise a particular organism or are they intrinsic only to a restricted class of cell population? Given a cell population in which there are basic mechanisms that are intrinsic, is there only one kind of mechanism of aging which is responsible for the broad array of features which characterize the elderly? Or are there a number of different kinds of aging mechanisms? The biologists have not begun to answer these questions, and in many cases, they have not even begun to ask these questions.

What about a general experimental approach that is capable of answering or resolving these particular issues? What might we consider as reliable, reproducible parameters of aging? There are many parameters which correlate with the passage of chronological time as members of a population approach their apparent maximal life span. For example, elderly human populations are characterized by increased susceptibility to most diseases, diminished organ function, altered metabolism, etc. (*1, 2*). Similarly, there are a few parameters which correlate with apparent maximal life span of specific populations. For example, maximal life span of at least certain species may correlate with the capability for

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repair of a specific type of DNA damage (*3*), with the susceptibility of DNA to chemically induced mutagenesis (*4*), and with the likelihood for proliferation and survival of specific cell populations grown in culture (*5*).

The current state of the art is primarily descriptive. This is not a criticism, it simply reflects the embryonic state of the field. Having documented a series of reliable parameters of aging, the next question lies in what cell populations of what particular tissue will such a lesion originate? Once you have identified a particular cell population, what is the limiting molecular event whose modification is responsible for this specific lesion in question? Having identified a specific biochemical event, what is the developmental profile of this lesion? At what age in the life span of the animal in question is this lesion of aging first expressed? And what is the nature of those factors which are responsible for its initial appearance? Finally, how do these factors relate to potential health and longevity and what types of methodology can be developed for their successful intervention?

Clouding the interpretation of even the reliable information is the inability, thus far, to ascertain the relative importance of contributions to the phenomenology of aging by genetic and environmental factors. One feature which probably characterizes all aging populations is the progressive modified ability to adapt to changes in the surrounding environment. One biochemical expression of this manifestation of aging is the altered capacity for the production of key enzyme molecules in response to some type of environmental challenge.

For example, the ability to initiate adaptive increases in the activities or amounts of nearly 50 enzymes now is known to be impaired during aging in a variety of tissues of several different species and in response to a broad spectrum of nutritional, pharmacological and environmental stimuli. These patterns of age dependent enzyme adaptations are susceptible to considerable variations; variations which are related at the very least to differences of sex, strain, species, growth, obesity, susceptibility to disease, as well as a host of other environmental interactions in the experimental population in question.

One clear-cut option now available to the research biologist is the use of inbred strains of experimental animal models which are maintained under rigorously defined and controlled environmental conditions (e.g., *6*). The clinical researcher, on the other hand, must face up to the consequences of the unfortunate reality that genetic and environmental influence on groups of human patients almost is beyond control. The spectrum of altered adaptive mechanisms is extremely diverse, ranging from the lessened ability of an elderly pedestrian to avoid an oncoming car to the impaired molecular recognition of a specific hormonal signal by a target cell population (*7, 8*). Accordingly, the epidemiologist should think in terms of which biological adaptations are most relevant to the question at hand, and what type of practical testing procedure can be devised in order to assess responsiveness conveniently in large populations.

Now let me show you one particular example of an age dependent enzyme adaptation which has occupied a great deal of attention in our laboratory during the last several years. Following administration of glucose to fasted, 2-month-old male or female Sprague-Dawley rats, the activity or amount of the liver enzyme, glucokinase, increases according to the indicated time course (see figure I-3). When the identical experiment is performed in older animals, in this case 24-month-old

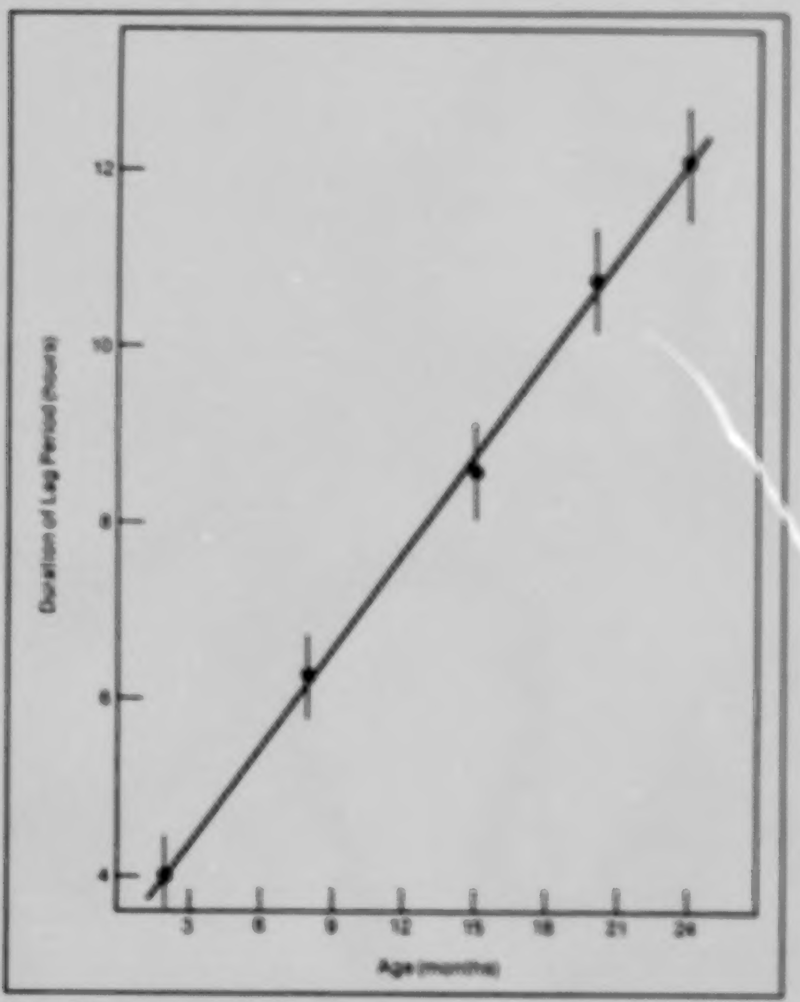

Figure I-8.

Relationship between chronological age of the rat and the duration of the lag period of the glucokinase induction. (Reference 9).

rats, more time is required to adapt to the same degree. In figure I-4, the time which elapses between the administration of the inducing stimulus, glucose in this case, and the observed initiation of increased enzyme activity, increases progressively during aging and actually is directly proportional to chronological age between 2 and at least 24 months. Similar age-related changes are shown for adaptations of tyrosine aminotransferase in response to ACTH and of cytochrome reductase in response to phenobarbital (8, 10).

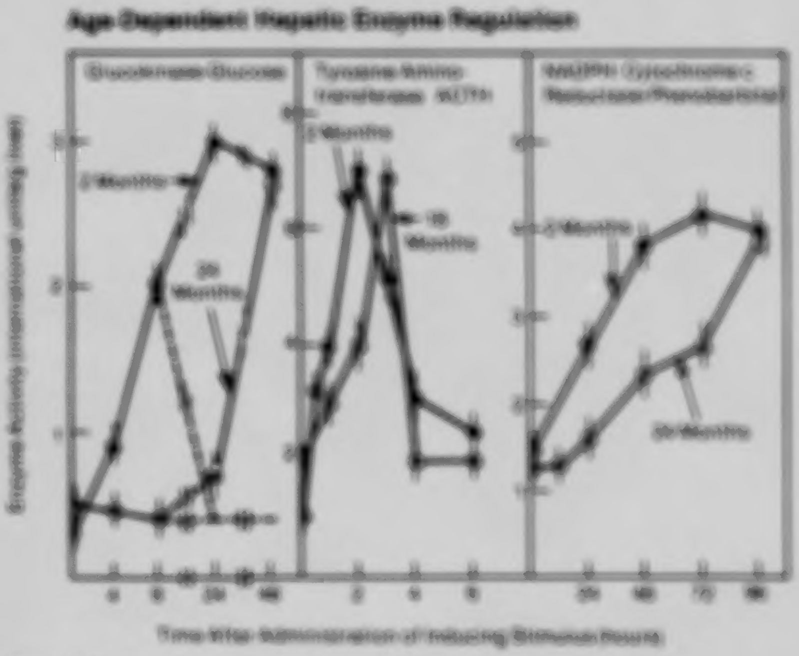

Figure I-4.
Age-dependent hepatic enzyme regulation. Each value represents the mean ± standard error for at least six rats. (Reference (8)).

This type of relationship now has been established for large numbers of different enzymes. Several years ago, we proposed this type of observation as the first biochemical parameter of aging. Because these measurements were performed in hepatic tissues, does this necessarily mean that the lesions responsible for changes in enzyme regulation are intrinsic to the liver? Our work has demonstrated that the lesions are not intrinsic to the liver itself. Instead they reflect a variety of endocrine disturbances, one of which refers to an age dependent lesion in the glucose stimulated control of insulin production. There is an age-dependent change in the control of insulin secretion such that the time of onset of a portion of the burst of insulin secretion also is delayed. This could account for the age-dependent change in the control of hepatic glucokinase activity, although the precise mechanism is not understood.

In conclusion, it is clear that there are a number of biological parameters of aging which are very easy to collect and accumulate in a variety of different experiments. In order to be absolutely certain of their reproducibility in the laboratory, the key feature is a rigorous definition of the experimental population. One of the most reliable parameters of biological aging clearly is the altered capability for adaptation. Adaptation to a variety of physiological challenges alters during aging in ways that are not so apparent when testing only resting functions or levels. Accordingly, as reliable parameters for epidemiological testing, my most enthusiastic recommendations include such things as altered response to administration of hormones, drugs, nutrients, and so forth; altered immune response to specific antigens; altered physiological response to exercise and other conditions of stress.

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Genetic and Immunologic Determinants of Aging*

Marc E. Weksler, M.D.

Department of Medicine
Cornell University Medical College
The New York Hospital
1300 York Avenue
New York, NY 10021

Since ancient times, longevity has been ascribed as a characteristic of certain human family and of [illegible] Despite the obvious influence of the environment on the attainment of maximal life span, the length of life is defined by the genetic constitution of the organism. Genes control longevity by regulating maximal life span of the species, and by regulating those biologic-defense mechanisms which are necessary for the organism to attain maximal life span.

Genetic Determinants of Life Span

This concept is [illegible] present evidence that genetic control of life span is determined by two biological processes:

(1) The duration and number of cell cycles that somatic cells can attain; and

(2) The duration of normal immunologic function.

Little experimental data are available about the genetic control of the maximal life span characteristic of a given organism. A theoretic hypothesis offered to account for the finite life span of an organism is that proposed by Hayflick (1). This hypothesis in effect suggests that life span is determined by the span and rate of cell cycles that can be carried out by somatic cells. When the number of proliferative cycles of a critical cell type are completed, senescence (death) has been attained.

Considerable evidence is available for the genetic control of biological defense mechanisms. Durability of these mechanisms is critical if the maximal life span of an organism is to be reached. If resistance to arteriosclerosis and neoplastic diseases, major factors in long life, is weak, life is usually shortened.

Resistance to such scourges as [illegible] is genetically inherited. An important determinant of human arteriosclerotic cardiovascular disease is the level of cholesterol synthesis. Cholesterol synthesis is largely determined by the expression of genetically specified cell-surface lipoprotein receptors (2). Excessive cholesterol synthesis occurs when cells fail to express these surface receptors for lipoproteins.

Similarly, the susceptibility of experimental animals and man to neoplasia is largely determined by the genetic makeup of the organism. The resistance to spontaneous or viral neoplasia in experimental animals is related to the histocompatibility complex and is

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Immune Reactivity and Genetic Control of Longevity

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[illegible]

Another method to establish a causal relationship between impaired immune function and aging is to alter immune reactivity and determine the effect on life span. Two environmental modifications, diet and body temperature, influence immune reactivity and life span. Caloric restriction without malnutrition markedly prolongs the life span of rats [illegible] and mice [illegible]. Dietary modification also has prevented the premature autoimmune disease and death of NZB mice [illegible]. Life span has been prolonged in fish by reducing body temperature. Attempts to lower temperature of homeotherms have been unsuccessful [illegible].

Age-Related Defects in Immune Reactivity

The senescence of the immune system affects both humoral and cell-mediated immunity [illegible]. Critical analysis of the multiple immune defects found in aged experimental animals and man strongly suggests that the involution of the thymus and consequent decline in T-lymphocyte function underlies the waning of immune function. Impaired rejection of grafts, impaired graft-versus-host reactions, increased susceptibility to neoplasms, and depressed proliferative and cytolytic reactivity of lymphocytes found in aged subjects all reflect faulty T-lymphocyte function. Humoral immunity, a direct expression of B-cell function, is also impaired during aging. However, the B-cell responses usually assayed are T-cell dependent. Thus, the impairment found may result from defective T-helper cell function and not from defective B-cell function. B-lymphocyte functions which do not depend upon T-cells are relatively well preserved throughout life [illegible]. Thus, responses to T-independent antigens or mitogens are only modestly impaired in old animals (11).

We have studied the humoral response of old and young mice to dinitrophenylated bovine gammaglobulin (DNP-BGG) (17). The B-cell response to this antigen is highly T-cell dependent. When the anti-DNP plaque forming cell (PFC) response of spleens from old and young mice immunized with DNP-BGG are compared, old animals show a preferential loss of IgG and high avidity PFC. Generation of IgG and high avidity antibody depends on normal T-cell function [illegible]. Thus, the impaired response of old mice to DNP-BGG in general and the preferential loss of IgG and high avidity PFC specifically point to defective T-helper cells in old animals. This interpretation is supported by the ability of young thymus cells to reconstitute the capacity of aged spleen cells to form IgG and high avidity PFC. We have recently been able to reverse the loss of high avidity PFC and augment the IgG PFC response of old mice by treating old mice with thymopoietin for [illegible] weeks prior to and [illegible] weeks following immunization [illegible]. The impaired high avidity and IgG PFC response of spleen cells from aged animals can also be reversed by incubating aged spleen cells with thymopoietin in vitro prior to their transfer to young irradiated thymectomized syngeneic recipients. In summary, the impaired antibody response of aged mice to the DNP determinant is very likely explained by the loss of effective helper T-cell function which results from a loss of thymic endocrine function.

One of the first studies of human T-lymphocyte function in aged subjects was the report of [illegible], et al. ([illegible]). These investigators found that the proliferative response of human lymphocytes stimulated by the T-lymphocyte dependent mitogen, PHA, was inversely proportional to the age of lymphocyte donor. We have studied the cytokinetic basis of this age-associated defect of lymphocyte response ([illegible]). Initial studies confirmed the impaired proliferative response of T-lymphocytes from older persons to plant lectins. This was not due to a decreased percentage of T-lymphocytes in the lymphocyte preparation from old persons. Furthermore, the age-associated defect in lymphocyte response to mitogens was demonstrable even when purified T-lymphocyte preparations from old persons were used.

The total proliferative response of lymphocytes cultured with mitogen depends on the number of lectin-responsive lymphocytes in the lymphocyte preparation and the capacity of their progeny or other lymphocytes recruited by them to enter the proliferating pool of cells. These two components of the lymphocyte response to mitogens may be dissociated by estimating independently the number of initially responsive lymphocytes and the number of second and third generation responding cells. Although the percentage of blood T-lymphocytes was not reduced in old persons, the number of mitogen-responsive T-lymphocytes was reduced. Three independent assays were used to estimate the number of mitogen-responsive cells in lymphocyte preparations from old and young persons. Limiting dilution analyses and colchicine block studies indicated that lymphocyte preparations from persons over [illegible] years of age contained approximately one-half the number of mitogen-responsive cells found in lymphocyte preparations from persons less than [illegible] years old. The third technique used to enumerate the number of lymphocytes activated by mitogens was the vesicular stomatitis virus plaque assay. This assay revealed that lymphocyte preparations from older persons contained one-fifth the number of mitogen-activatable lymphocytes found in lymphocyte preparations from young persons. Thus, each of these three assays demonstrated that the number of mitogen-responsive lymphocytes was reduced in older persons.

The first clue that the progeny of mitogen-responsive cells from old or young persons do not proliferate equally in cultures came from our studies in which the amount of thymidine incorporation in the absence or presence of colchicine was compared. In the presence of colchicine, only first generation responding lymphocytes incorporate thymidine. In the absence of colchicine, the entire pool of proliferating lymphocytes incorporate thymidine. Thymidine incorporation by cultures from old persons was relatively more impaired in the absence of colchicine than in the presence of colchicine. This suggested that cells from old persons failed to expand into a clone of proliferating cells. This defect was documented by two types of experiments. In the first, colchicine block and thymidine pulse studies were conducted. This technique revealed two peaks of thymidine incorporation in lymphocyte cultures from young persons, one occurring on the third day of culture and the second on the sixth day of culture. The latter peak represents a second or third round of lymphocyte proliferation. This peak did not occur in cultures of lymphocytes from old persons. The second experiment measured thymidine incorporation by increasing numbers of cells placed in culture. When the number of young cells in culture was increased arithmetically, the amount of thymidine incorporated increased exponentially. In contrast, there was a linear increase in thymidine incorporation by cultures from old persons with increasing numbers of cells. Lymphocytes from young persons cultured in the presence of colchicine showed a linear rise in thymidine incorporation as the number of cells in culture was increased. This demonstrated that the exponential increase in thymidine incorporation was due to entry of progeny cells into the pool of proliferating cells. Thus, the impaired response of lymphocytes from old persons results from two distinct defects. One defect is the reduced number of mitogen-responsive lymphocytes already well-documented. The second defect is a failure of mitogen-responsive lymphocytes to expand into a clone of proliferating cells.

As thymic hormone concentration in serum falls with age (26), we tested the effect of thymopoietin on the impaired response to mitogen by lymphocytes from old persons. Thymopoietin increased the amount of thymidine incorporated by lymphocytes from old but not young persons in the presence of colchicine. This suggests that thymopoietin increases the number of first generation responding cells in lymphocyte preparations from old subjects. Thymopoietin had no effect on the recruitment of progeny cells into the proliferating pool of cells (27). These results suggest that the loss of mitogen-responsive lymphocytes is a consequence of the senescence of thymic gland function and that thymic hormone converts mitogen-unresponsive cells to mitogen responsive cells. The failure of thymopoietin to increase clonal expansion is not surprising as this defect reflects the exhausted reproductive capacity of somatic cells from older persons.

A model of the two factors which contribute to the age-associated defect in mitogen-induced proliferation is illustrated in figure 1-3. The figure compares the response of T-lymphocytes from young and old donors to mitogens. All the T-lymphocytes from young persons are shown to be mitogen-responsive while only three out of five T-lymphocytes from old persons are responsive. Nonresponsive lymphocytes can be made mitogen-responsive by thymopoietin. The total proliferative response of lymphocyte cultures from young individuals depends on the proliferation of second and third generation cells. Second generation lymphocytes induced in cultures from old persons are not stimulated in culture and thymopoietin does not influence this defect.

These studies reveal two genetic factors that determine human life span which are probably linked to the histocompatibility complex.

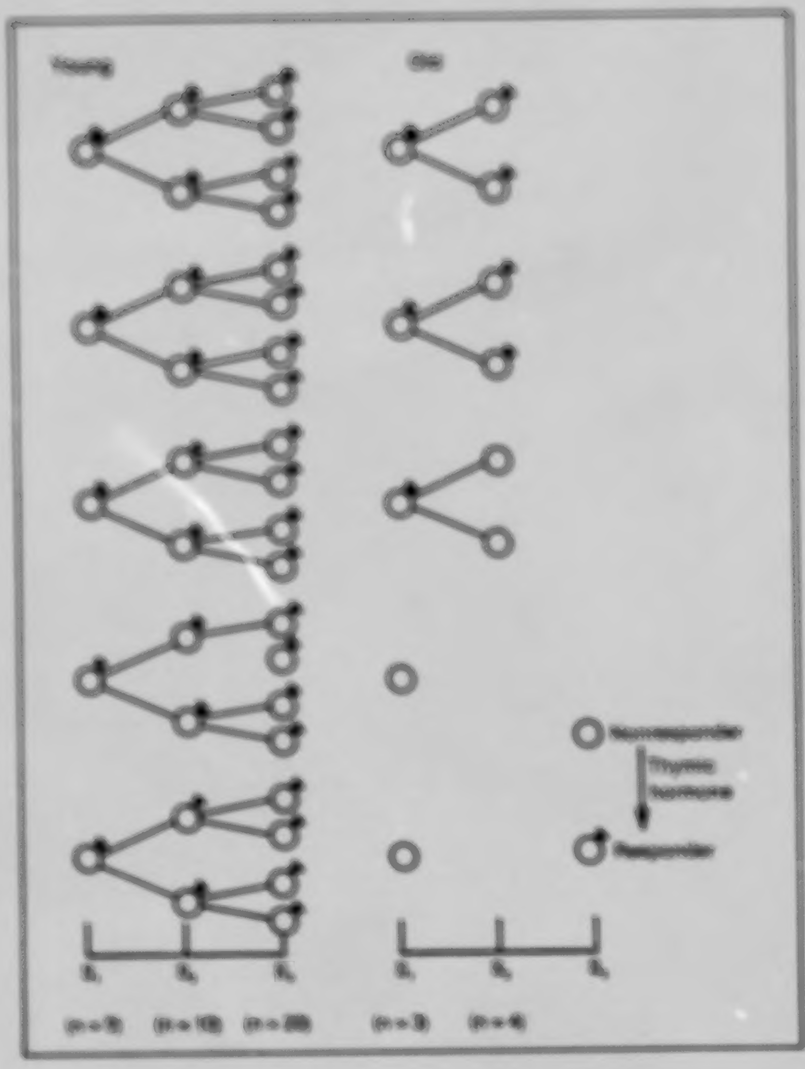
Young
Old
Nonresponder
Thymic hormone
Responder
G_1
G_2
G_3
G_1
G_2
G_3
(n = 5)
(n = 10)
(n = 20)
(n = 3)
(n = 4)

Figure 2.4.
Model of the age-associated defect in thymidine incorporation by lymphocytes in culture.

One factor is the proliferative capacity of somatic cells. The second factor specific for the immune system is a loss of thymic endocrine function. This, of course, has implications beyond the immune system for the age-associated disorganization of the immune system comprises several biological defense mechanisms. What are the implications of these findings? Certainly the rejuvenating effect of thymopoietin on T-lymphocyte function is encouraging. Whether this will allow subjects to achieve maximal life span more frequently must be tested experimentally. Means to extend the proliferative limitations of somatic cells seem more distant.

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Discussion

Waldron: With regard to responsive cells, did you feel that there were fewer cells or that fewer of the cells were capable of responding?

Weksler: It is probable that both are correct. First, fewer cells are capable of responding and second, those that do respond do not proliferate normally. Whether these observations are related is another question. We have two observations which we seek to relate.

Ostfeld: Have there been any lymphocyte studies of the kind you mentioned made either in young diabetics or in young people with accelerated atherosclerosis?

Weksler: No.

Ostfeld: Would you say that these changes may represent the kinds of lymphocytes that exist in people who survive or who are relatively immune to atherosclerosis, hypertension, or diabetes, rather than to some process called aging that we can separate from it?

Weksler: That is really one of my points. The survival influence of immunity on aging may well be expressed in those terms. Persons who live to 80 may have greater immune competence which is protective. If immune competence exists, the evolutionary pressure must be exerted earlier in life. It may be that operationally we cannot see it as clearly because of the regression of the mean or for other reasons. The statistics are such that the differences are much smaller at that point and are only magnified by age, but the critical biological defense mechanism may be exerted in midlife.

Functional Age: A Conceptual and Empirical Critique*

Paul T. Costa, Jr., Ph. D. and Robert R. McCrae, Ph. D.

Gerontology Research Center
National Institute on Aging
Baltimore City Hospital
Baltimore, MD 20224

Scientific evidence and common sense concur in seeing age as a powerful variable for ordering information about the status and functioning of organisms. Social and biological age stereotypes are among the most important folk concepts for understanding people, while the existence of the field of gerontology testifies to the scientific utility of using chronological age as a mediating and ordering variable. Aging, whether it is seen as a controlled or random process (*1*), determined by genes, environment, or accumulated trauma and pathogenesis (*2*), affects a wide variety of biological, psychological, and social processes, and all of these, in turn, can be roughly indexed by chronological age.

But on a little reflection, it becomes clear that chronological age is not in itself an explanatory variable, but rather a kind of index, backdrop, or dummy variable which is used to stand for the process or processes which causally underlie the universal, progressive and deleterious changes which we call aging. It is equally clear that chronological age is at best a rough approximation to these changes and that some individuals appear to be younger or older than their years in various respects. Folk wisdom notes that you are "as young as you feel," or prematurely aged. Scientific data point to the same phenomenon in noting the range of individual differences in performance on age-related functions for any single age cohort.

Functional age is one of a family of concepts which have been introduced in an attempt to take these deviations from a simple chronological age model into account, to produce an alternative to chronological age which is of equal generality and greater precision. In some respects functional age is an attempt to find the gerontological complement to the concept of developmental age. In the form of mental age, the numerator in Stern's famous equation for the intelligence quotient (IQ), this concept showed considerable utility and validity, though it has now been superseded by more sophisticated conceptualizations. "Mental age" worked as well as it did because in the first 16 years of life, the performance on intellectual tasks is so well ordered by chronological age that individual differences can conveniently be considered as differences in mental age. The idea of mental age was ultimately abandoned because individual differences remain after adulthood, while intelligence in adulthood shows little relation to age. These considerations about the relative importance of individual variation and degree of age-relatedness will be seen to be crucial to an evaluation of functional age.

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Within gerontology itself, a number of precursors of the functional age concept can be identified. Benjamin (*3*) noted variation in the condition of body organs at a given time which resembled the variation introduced by age, and proposed the idea of "organ age." In 1959, Birren (*4*) offered a conceptualization of the aging process in terms of a hierarchy of biological, psychological, and social ages. Heron and Chown (*5*) identified eight variables which showed a linear relation with chronological age and created a functional age profile on these eight separate measures: FEV, grip strength, sitting height, low nonverbal intelligence, poor perceptual maze performance, poor digit coding, systolic blood pressure, and hearing loss. The authors did not combine these measures to form a functional age score in years because they felt the diversity to be too great and the statistical correlation insufficient. Comfort (*6*) proposed measuring a variety of biological and psychological characteristics to gauge the age of the individual. At the Elkridge Conference on the Epidemiology of Aging (1972) the question was raised for future investigators as to whether Comfort's measures can provide a useful index of aging. They were: hair greying, height, seated height, trunk height, biacromial diameter, skin thickness and elasticity, thorax size, systolic blood pressure, diastolic blood pressure, total vital capacity, tidal value, FEV_1. The term "functional age" was introduced in the 1950's by McFarland (*7*), who also did some pilot work on aviators. The major studies of functional age, to be reviewed below, were performed by Dirken (*8*), who attempted to assess the functional age of Dutch industrial workers; and by workers at the Normative Aging Study (*9–11*) who developed six indices of functional age for a population of initially healthy adult males. Furukawa et al. (*12*) in their studies to assess what they labeled "biological age" used the same procedures and rationale in a Japanese sample (see figure I–6).

The concept of functional age which has been developed has been alternatively phrased, but seems to rest primarily on the notion of differential rates of aging in individuals. Building on the appealing commonsense idea that some people "burn themselves out" rapidly while others are "well preserved," and on the observation of individual differences in longevity, this formulation has postulated that the variation of performance around a cohort mean is due to the different rates of aging of individuals. At any given point in time, those who age rapidly will be closer to death than those who age slowly, and they will, on the average, perform less well on age-related measures of functioning. These rapidly aging individuals will be "functionally old" in comparison with their chronological age peers.

While most investigators have considered the possibility that different systems age at different rates within the same individual, so

Normative Aging Study	Webster & Logie	Furukawa et al.	Dirken
Greyness of hair	Plasma urea nitrogen	Glomerular filtration rate	Resting EKG
Disassemble (GATB)	$\log_{10}$ alkaline phosphatase	Renal plasma flow	Exercise EKG
Speech reception loss	Erythrocyte sedimentation rate	Systolic blood pressure	Maximum energetic load
FEV_1	Plasma triglycerides	Diastolic blood pressure	Maximum pulse frequency
Length of ear	Plasma cholesterol	Cholesterol	Maximum system blood pressure
% hemoglobin	Systolic blood pressure	Red blood cells	VC_1
Chances for advancement	FEV_1	White blood cells	FEV_1
Perceived position in company		Hemoglobin concentration	Maximum breathing rate
Surgency		Icterus Index	Urinary 17-oxogenic
Expected age of retirement		Total protein	17-oxosteroids
Plans to remain with same company		Height	Creatinine
Hearing loss for 8 KHz tone		Weight	Psychomotor positioning
		Vital capacity	Tapping rate
		Ocular accomodation	Hand dynamometry
		Vibratory sensation	Speech audiometry
		Grasping power	Pitch ceiling
		Tapping rate	Presbycusis
		Body flexibility	Visual acuity
		Heart rate recovery	Picture completion
			Reaction time
			Concentration

Figure I-4.
Variables used in functional age prediction equations in four studies.

that, properly speaking, everyone would have a number of different functional ages, in practice measures from a variety of domains have been combined to produce a single prediction of functional age. The various indices of functional age identified by the Normative Aging Study were later combined (*13*) to form a single index. And such a combination is understandable: part of the appeal of the notion of functional age is that it will provide an accurate but practical alternative to chronological age. To be practical, any proposed alternative must share the wide generality of chronological age, so that a multitude of functional ages would defeat one of the chief purposes of the concept. This paper is intended as a critique of functional age concepts which aspire to such generality; other concepts, limited to single organs or functions, are not considered here.

Implicit in these attempts to generate an age assessment from a variety of age-related performance measures is the (questionable) notion that a single aging process underlies all the changes associated with the passage of time. Examples can be cited which fortify this position: mental status often depends on the condition of the arteries, while social status will often depend on mental performance. The adage propounded by Ostfeld, that "you are as old as your arteries," expresses this position neatly. If this assumption is true, or even approximately true, then the combination of measures of a variety of performances which share in common their manifestation of the common aging process seems to be a plausible approach to finding an alternative to chronological age.

Functional age as a focus for gerontological research has both practical and theoretical appeal, and numerous claims have been made for it by its exponents. Dirken (*8*) and McFarland (*7, 14*) have been concerned with problems of social policy, and in particular, retirement. Making retirement mandatory at age 65 seems inappropriate since many individuals can continue functioning well in their jobs long past this age, while others show signs of incompetence years before. A single measure of their ability to function (or "functional age") would, these writers contend, provide a more appropriate criterion for retirement.

Another use of functional age would be the study of various environmental impacts on the aging process. Longevity research as currently conducted deals with this issue in such areas as comparing urban and rural environments in terms of differential life expectancies. The unavoidable drawback of longevity research is the necessity of waiting until individuals have died. Functional age is believed to promise an alternative in which the effects of life conditions can be assessed at any point in the life cycle by comparing the functional age of various groups. This should both speed scientific progress and allow interventions which will be of use to the individuals studied.

Finally, functional age might provide a much more sensitive index of aging in the individual than chronological age does, and this in turn might make study of the processes of aging easier. Currently most research on aging is done cross-sectionally, rather than longitudinally.

Despite the limitations of cross-sectional research, its economy will make it a continuing approach for gerontological research for the foreseeable future. If, however, chronological age is only a rough guide to "true" age, then these studies will suffer from some unknown degree of error. If a meaningful functional age could be obtained, *functional age cohorts* rather than chronological age cohorts might be used in cross-sectional research, with the result that relationships might be much clearer. By shifting from a geocentric to a heliocentric frame of reference, the course of the planets became understandable; by shifting from chronological age to functional age equal clarification of the course of human life might be expected.

The origins of the concept of functional age in common sense and scientific observation, and the practical and theoretical possibilities of the idea have been spelled out in detail to explain the enormous appeal of the idea. A clear understanding of that appeal seems necessary both in order to understand why attempts to measure functional age have so often been made and to anticipate the inevitable disappointment which will follow from a critical evaluation of the concept and its supporting evidence. For despite the appeal, despite the wishful thinking that inspired claims for functional age, there is presently no evidence for utility in the concept. Both logic and hard data belie the apparent underpinnings of the concept, and alternative methods of dealing with the problems functional age is supposed to solve can be proposed. Rather than "Keplerian," the functional age concept will be shown to be "Ptolemaic" in nature: an early, prescientific approach which showed considerable promise and occupied the efforts of many distinguished thinkers, but which would be best abandoned.

Studies of Functional Age

The first and most elaborate study of functional age using the multiple regression model was conducted by Dirken and his colleagues in the Netherlands. As industrial gerontologists, they were concerned with finding an alternative to chronological age as a basis for retirement policy.

In their attempt to develop a "yardstick" of aging, they examined 150 variables from psychomotor, sensory, and biomedical domains on a sample of 336 Dutch workers between 30 and 70. Elaborate analyses were conducted, including the calculation of age-corrections for regression to the mean. As with most other functional age studies, the ratio of variables to subjects was quite large, allowing considerable influence of error. Neither external criteria nor longitudinal followups were used to validate the predictions.

A series of studies at the Normative Aging Study (8-10) attempted to define functional age from measurements in six domains on a population of 2,000 adult males initially selected for health. Functional age equations were generated from anthropometric, social, auditory, laboratory, personality and ability measures. The squared multiple correlations for the different domains, indicating the percent of variance in chronological age which could be predicted: domain: social, [illegible]; anthropometry, [illegible]; abilities and personality, [illegible]; and audiology, [illegible].

An attempt was made to determine whether individuals showing extreme deviation of functional age from chronological age in one domain also showed deviations in others ([illegible]). If so, it would provide evidence for a general rate of aging. No statistical evaluation of the results was made, however, so interpretation is unclear.

At a later date, Fozard and Thomas ([illegible]) combined the variables from all six domains in a single regression equation predicting chronological age. No validation of the resulting equation was attempted. The major predictors are listed in figure [illegible].

Furukawa et al. ([illegible]) tried to assess "biological age" by regressing a number of biomedical factors onto chronological age. Four separate studies were conducted: on [illegible] normal subjects, on [illegible] employees and students at a medical school, on [illegible] male and female hypertensive patients, and on [illegible] healthy employees. The variables entering into the regression are given in figure [illegible].

One novel feature of this study was the comparison of normal and clinical samples. When prediction equations developed on normotensive subjects were applied to hypertensives, the predicted age was higher than chronological age. It might appear that hypertensives are functionally older than normals. An equally plausible interpretation would note that many of the same functions which are sensitive to aging are sensitive to disease also, and therefore it could be concluded that hypertensives are merely sicker than normals.

Webster and Logie ([illegible]) followed the usual rationale for predicting functional age from seven clinical variables in a sample of [illegible] apparently healthy female subjects aged [illegible] to [illegible]. However, these investigators took the additional step of attempting to validate the prediction. A subsample of [illegible] nonsmokers selected for a history of health were shown to have a mean predicted age less than chronological age by [illegible] years. Further, when these subjects were subdivided according to subjective or perceived health, those with best perceived health were functionally younger.

While problems remain in this study in the conceptual confounding of aging with disease and in the failure to deal with regression to the mean, the attempt to predict external criteria makes this study perhaps the only evidence for the validity of functional age in the literature.

Examination of figure [illegible] shows that there is only small overlap between studies in the measures used to estimate functional age. Since different initial sets of predictors were used, the studies cannot be considered replications of each other, and the degree to which prediction equations were influenced by chance remains unknown.

A few variables—FEV1, blood pressure, hearing loss—are common to two or more studies. This provides evidence of their consistent relation to age, but does nothing in itself to demonstrate their ability to assess functional age.

[illegible]

[illegible]

Critique of the Functional Age Concept

[illegible]

[illegible]

[illegible]

[illegible] and below. This model can be criticized on several grounds: (1) multiple regression is not an appropriate method; (2) chronological age is an inappropriate criterion; (3) interpretation of the results is implausible; and (4) validating evidence has not been offered.

Multiple regression is a technique for assigning weights to a set of variables which will maximize the correlation of the resulting linear combination with some criterion variable. Beyond the usual and usually disregarded assumptions that all the variables are in ratio scale and have a joint normal distribution, it is assumed that the relations between the predictors and criterion are linear. In fact, many of the variables used show different rates and curves of age-related decline. The practical significance of this fact is that some variables are inappropriate for predicting functional age in some age groups. When separate regressions are run for the young and old cohorts (see table 3.1) it can be seen that age is only weakly related to "age-related" variables in the young group, while stronger relations are seen in the old group. Table 3.1 shows that, for example, card sorting, a test of perceptual ability, is unrelated to age among young men. To attempt to assess their functional age on the basis of their scores on this variable is absurd, yet it is an inevitable consequence of the approach which has been used.

Chronological age has been selected as the criterion for regression analysis because it was always available, and because it was assumed that the mean functional age for any cohort was given by the chronological age. If this is intended as part of the definition of functional age, such an assumption is legitimate but somewhat tautological. This makes some sense if we intend to define functional age in these terms, that is, if we mean by functional age the level of performance on an age-related task which characterizes an age cohort. This is the sense in which we say that a man has "the heart of a 30-year-old," even though he is 60.

However, the usual conception of functional age is in terms of a rate of aging distinct from chronological age ([illegible]). Functional age then must be a reflection of the rate at which a person ages. Chronological age is an appropriate criterion for regression only if it can reasonably be assumed that the average rate of aging is given by the passage of calendar time. This, in turn, means that aging proceeds at a uniform rate during the adult years, an assumption which is contradicted by any number of studies. If a single generality were to be propounded, it would be that the decrements in performance accelerate with age. This can be seen in mortality, deafness, frequency of illness, and many other age-related processes. It is patently inappropriate to use as the criterion of an accelerating process ("functional aging") a variable which is linear ("chronological aging").

Further, there is a kind of circularity in using chronological age as the criterion for developing an alternative to chronological age. To be sure, variables which index aging must show changes with the passage of time, and in the absence of longitudinal evidence, cross-sectional correlations with chronological age can be used to indicate this. How-

Table 5-6.
Regressions predicting chronological age in young and old groups

	Young					Old				
	Simple r	Beta	Mean	S.D.	N	Simple r	Beta	Mean	S.D.	N
C. age	—	—	[illegible]	[illegible]	[illegible]	—	—	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Speech reception threshold	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Ear length	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
		Multiple R: [illegible]					Multiple R: [illegible]			

over, the logic of combining age-related variables in such a way as to maximize the correlation with chronological age seems pointless. If the regression succeeded perfectly, the resultant statistical age would correlate 1.0 with chronological age, and hence would be a perfect, and perfectly useless, alternative to chronological age.

The statistical ages produced by regression do not in fact correlate perfectly with chronological age. Because of this, there is a discrepancy between the predicted and the actual age of each individual which can be used to classify him/her as functionally young or old.

It is assumed that where statistical age is higher than chronological age, the individual is functionally old; where statistical age is lower, the individual is functionally younger than his peers. Because of the statistical artifact of regression to the mean, it will necessarily be the case that old men will tend to have younger predicted ages, while young men will have mostly older predicted ages. Only for men near the average age is the regression equation a very meaningful candidate for the title "functional age."

Even here, the assumption is made that all deviation about the cohort mean is due to differential rates of aging; that those who perform worse are those who have aged more rapidly than their peers. In fact, however, it seems plausible, as Dempster (22) points out, that deviation around the mean will be the result of several sources of variance, including innate individual differences in level performance, short-term variation in the variables measured, and error of measurement (and in cross-sectional studies, cohort difference). The assessment of functional age is possible in these circumstances only if the proportion of variance due to these other factors is small in comparison to the variance introduced by differential rate of aging. The concept of mental age was abandoned when individual differences were seen to outweigh developmental differences in adulthood; functional age will be useful only if functional aging differences are more powerful than all these other sources of variance, and that is, a priori, very implausible.

Day-to-day variation in physiological measurements like blood pressure and glucose levels are notoriously high, as are errors of measurement in psychological measures. The importance of individual differences in initial level is seen, for example, in the fact that chest circumference, a presumably genetically determined variable, is a much better predictor of vital capacity than age. In the face of these known sources of variance, the assumption that the deviations between predicted and chronological age represent the effects of differential rates of aging seems gratuitous.

However, the test of a scientific proposition is not in the prior plausibility of it, but in its predictive value. And the greatest shortcoming of the functional age studies to date is their failure to provide convincing evidence that they are measuring what they claim to be. Longitudinal research is clearly needed to verify or falsify these propositions, and a definitive answer would require an extensive commitment of research time and effort. In view of the conceptual flaws in the notion of functional age such work may not be justified. However, in order to give the approach the benefit of the doubt, several analyses using longitudinal data from the Normative Aging Study were performed. None of these can be considered a definitive test of the concept. The criteria are not optimal, the time spans are too short, and the analyses suffer from some limitations of time. Nevertheless, as the only longitudinal evidence directly bearing on functional age, they should be useful indicators of whether the approach is or is not worth pursuing.

Testing the Validity of Functional Age

The functional age formulas derived by investigators at the Normative Aging Study were derived from the first cycle of data collected there. Since then, a full second cycle and most of a third cycle have been collected, and it is therefore possible to examine the validity of functional age formulas in predicting longitudinal changes. Given the six sets of predictors and the numerous age-related variables which might be predicted, the following analyses are only a selection of the possible ones, but they should be useful in forming a tentative evaluation.

Four different kinds of analyses were performed to serve as alternative tests of the utility of the functional age concept. In the first, change in anthropometric variables was predicted from functionally older or younger status, to test the hypothesis that the functionally older would age more rapidly, and thus show greater changes. In the second, changes in a variety of performance measures over 5- and 10-year periods were predicted from a set of functional age indicators and from chronological age, again to test whether the functionally older showed higher rates of change. The third consisted of a factor analysis of the changes in all available age-related measures for two 5-year periods to determine whether a single factor of "rate of aging" could be identified; and the last returned to an examination of simple status at one point in time on age-related variables to see if some combination of age-related variables could out-predict chronological age. The analyses increased in sophistication as early results pointed to the need for readjustments. Doubtless, continued research will lead to more refinements, if any justification for continuing research can be found.

Predicting changes in anthropometric variables.—At any one point in time, it can be argued that individuals who age rapidly should perform, on the average, worse than those who age slowly. These two groups may be presumed equal in their distributions of initial levels; but, with the passage of time they should increasingly diverge. In any cohort, then, the individuals who perform better than average will include a disproportionate number of the slowly aging. It can be be expected that the better performers, the "functionally younger," will show less decrement over a fixed time interval than the "functionally older."

In order to test these assumptions, it would be necessary to classify individuals as "better" or "worse" than their peers at a given time. The variables selected to form the basis of this classification should be sufficiently sensitive to the effects of aging that it will discriminate between the rapidly aging and the slowly aging. Any single variable (such as greyness of hair) is likely to be relatively more sensitive to individual differences than to the aging process, and thus will be a poor classifying variable. A composite of variables which share in common chiefly their age-relatedness might be preferable, since individual differences unrelated to aging would tend to cancel. The six functional age equations offered by the Normative Aging Study provide such composites. Of these, the anthropometric formula for functional age seemed the most promising, since it was a particularly good predictor of chronological age (R=0.77) and since the variables used in anthropometric measurements, although showing changes small in absolute magnitude, are among the best for longitudinal research. Error of measurement is extremely small, particularly in comparison with, say psychological measurements; and day-to-day fluctuation in most variables is minimal, compared with changes in blood pressure or glucose. Because of this reliability, it has been possible to show significant longitudinal changes in many of these measurements (*25*), thus showing that they do indeed change over time and that observed cohort differences are not due solely to generational effects. In consequence, changes in anthropometric functioning should provide an extremely sensitive test of the proposition that individuals age at varying rates.

A series of two-way analyses of variance were performed in which the classifying variables were age group and relative functional age, and the dependent variables were changes in anthropometric variables known to be age-related. The assignment of individuals to functionally older and younger groups was made by subtracting the statistical or predicted functional age from chronological age, and grouping those with negative values as "functionally older"; those with positive values as "functionally younger" than their years. The prediction equation was that provided by Damon (*10*).

While the regression method assumes that aging is a linear process, much empirical data contradict this view, so that it seemed prudent to divide the subjects into three chronological age cohorts to see if rate of aging was related to chronological age, or if there were interactions between chronological age and relative functional age. Finally, in order to control for the initial level while examining change variables (*26*) the initial level of the dependent variables was entered as a covariate in each analysis.

Results, summarized in table I–2, show that while chronological age group was a significant main effect in several of the variables, relative functional age had no effect whatsoever. With subjects numbering more than 1,400, this seems highly damaging evidence to the claim that the functionally younger will change less.

However, it became apparent that the results might be due to a statistical artifact. Because of the use of multiple regression formulas in predicting functional age, regression to the mean must have occurred. That is, the chronologically oldest subjects are likely to have predicted values lower than their chronological ages and so be classified as "functionally younger," while the youngest subjects, for the same artifactual reason, are likely to be classified as functionally older. In fact, mean age of the functionally younger group was 50.3 years, mean age of the functionally older group was 39.2 years. Since the ANOVA's themselves show that rate of change is related to chronological age, the analysis confounds chronological age with functional age in such a way as to obscure the possible effects of the functional age classification.

Table I–2.

Analyses of variance of 5-year changes in anthropometric variables, functional status by cohort

Dependent variable	F for cohort	F for functional status	N
Change in:			
Baldness	1. 335	1. 185	1, 421
Greyness	16. 373 [2]	. 480	1, 416
Bideltoid	13. 892 [2]	. 152	1, 395
Abdominal depth	4. 573 [1]	. 363	1, 409
Triceps	1. 758	. 142	1, 422
Sitting height	19. 728 [2]	. 368	1, 394
Nose breadth	. 855	. 000	1, 424
Ear length	. 093	. 309	1, 423
Ear breadth	4. 275 [1]	2. 401	1, 423
Grip strength	27. 466 [2]	1. 275	1, 337

[1] $p = 0.05$

[2] $p = 0.001$

NOTE.—No interaction was significant.

On these grounds the analyses might have been omitted from this presentation. They are included, however, because they illustrate that the problem of regression to the mean, overlooked by Normative Aging Study investigators (among others), are sufficiently powerful as to vitiate the utility of the predictions over the whole of the age range. Relative performance within single age cohorts (based on perhaps 500 subjects per 5-year cohort) of variables known to change with age might be a useful way of classifying individuals as functionally young or old, but this approach requires extraordinarily large numbers of subjects, and has not been used by anyone to date.

Predicting changes from functional age indicators.—An alternative to the approach used above which circumvents the problem of regression to the mean relies on the use of functional age indicators to predict change. A set of highly age-related variables are used to predict change over 5- and 10-year periods, on the assumption that those who are high on these variables will show higher rates of change. For example, we might assume that grey-haired people, regardless of chronological age, will show more hearing loss over a 5-year interval than individuals with no grey hair.

Two problems must be settled before conducting such analyses. The set of functional age indicators must be selected, and the manner of their combination must be determined. Five variables were selected: greyness, ear length, speech reception threshod, the disassemble subtest of the GATB, which measures manual dexterity, and the tool matching subtest, which measures perceptual ability. These variables were chosen because they were among the best predictors of chronological age in Fozard's (*13*) regression; because they were relatively independent of each other except for the shared variance which was age-related; and because none of them cane from the biomedical domain, and were thus independent predictors of status in that most important area.

Rather than using a single estimate of functional age by combining these variables, it was decided that multiple regression would be allowed to select the best possible combination for each variable.

The criteria for these regressions were all the changes in variables known or presumed to be age-related which were available. In order to correct to some extent for differences in initial level, the changes were expressed as percent change over initial level. For example, forced expiratory volume (FEV) decreased from 3.52 to 3.01, or a loss of 0.51. This was expressed as a change of 14.7 percent from the initial level of 3.52 over 10 years.

The regressions were performed in two steps. On the first step, the five functional age predictors were entered; on the second step chronological age was added. Table I-3 shows the multiple R for the five predictors, the simple r for chronological age, and the combined multiple R using all six. Inspection of these values shows two things clearly: first, predictions are trivial in size and rarely reach statistical significance. No combination of these predictors can give much information about changes in any of the variables considered. Indeed, the largest R, predicting percent change in glucose, is only 0.249. Second, chronological age alone does almost as well as the combination of all six predictors in nearly every case. Inspection of the beta weights in the final step of the regressions show that chronological age is usually the best predictor. In short, chronological age (which the linear model adopted in functional age research assumes to be unrelated to rate of change) is the best predictor of what little change can be predicted, and little incremental validity is added by five of the best functional age indicators.

It might be objected that greyness, deafness, and so on are not in themselves indicators of rate of aging; they become so only in relation to chronological age. Being grey at 80 is not a sign of premature aging. Therefore, it is not to be expected that these measures will predict changes in other variables. A more appropriate model would consider these signs in relation to chronological age to provide a measure of rate of change. Controlling for chronological age would meet this *objection, and entering chronological* age into the regression equation is precisely equivalent to utilizing the best possible control for age. It does not, however, do much to improve the multiple correlations, so this objection is not sustained.

Table I–3.

Regressions predicting percent change in age-related variables, from functional age indicators and chronological age

Variable	df	Multiple R with 5 functional age indicators	Simple r with chronological age	Total multiple R
Baldness				
T_1–T_2	584	.091	.002	.097
T_2–T_3	250	.034	−.008	.051
T_1–T_3	201	.111	−.071	.123
Air Conduction, 8kc				
T_1–T_2	660	.039	.013	.045
Bideltoid				
T_1–T_2	660	.123	−.113[1]	.*150*[1]
T_2–T_3	250	.066	−.092	.133
T_1–T_3	261	.116	−.167[1]	.175
Abdominal				
T_1–T_2	660	.097	−.148[2]	.*153*[1]
T_2–T_3	250	.097	−.052	.123
T_1–T_3	257	.147	−.142[1]	.178
Triceps				
T_1–T_2	660	.097	−.063	.112
T_2–T_3	249	.152	.113	.155
T_1–T_3	258	.143	.098	.147
Sitting Height				
T_1–T_2	660	.*127*[1]	−.187[2]	.*200*[2]
T_2–T_3	250	.062	−.042	.068
T_1–T_3	259	.061	−.043	.068
Nose Breadth				
T_1–T_2	660	.064	−.036	.080
T_2–T_3	250	.137	.049	.138
T_1–T_3	249	.217	.015	.127
Grip Strength				
T_1–T_2	660	.093	−.062	.099
Cholesterol				
T_1–T_2	660	.076	−.003	.085
T_2–T_3	437	.032	−.044	.056
T_1–T_3	442	.076	−.000	.076
PBI				
T_1–T_2	660	.068	−.057	.073
T_2–T_3	429	.095	.021	.108
T_1–T_3	434	.145	−.023	.151

Continued

Variable	df	Multiple R with 5 functional age indicators	Simple r with chronological age	Total multiple R
Glucose				
T_1-T_2	660	*.147*[3]	.152[3]	*.166*[3]
T_2-T_3	440	*.229*[3]	−.223[3]	*.249*[3]
T_1-T_3	442	.118	.101	.139
SED				
T_1-T_2	660	.070	−.035	.072
T_2-T_3	439	.129	−.003	.127
T_1-T_3	442	.108	−.156[3]	*.187*[1]
Hemoglobin				
T_1-T_2	660	.107	−.063	.107
T_2-T_3	440	*.171*[1]	.052	*.178*[1]
T_1-T_3	442	.124	.032	.154
BUN				
T_2-T_3	347	.083	−.003	.083
FEV				
T_1-T_2	660	.056	−.060	.077
T_2-T_3	490	.075	−.003	.075
T_1-T_3	459	.139	−.183[3]	*.195*[3]
PVC				
T_1-T_2	660	.054	−.072	.091
T_2-T_3	490	.101	.087	.113
T_1-T_3	457	.094	−.008	.095
Systolic B.P.				
T_1-T_2	660	.058	.021	.058
T_2-T_3	367	.134	.113[1]	.161
T_1-T_3	369	.070	.092	.105
Diastolic B.P.				
T_1-T_2	660	.109	−.108[1]	*.138*[1]
T_2-T_3	367	.079	.036	.086
T_1-T_3	369	.094	−.047	.095

[1] $p=0.05$
[2] $p=0.01$
[3] $p=0.001$

Another objection is, however, valid. Aging, as most investigators have indicated, may be a nonuniform process. It may be that the cardiovascular system ages at a constant rate, but a rate unrelated to anthropometric or auditory declines. Such a possibility severely limits the utility of the functional age concept, but it may nevertheless be true. The set of predictors chosen may have been unfortunate; some other set not yet available may have shown many meaningful relations.

However, the criteria for selecting the "right" set is unknown. Some alternative to trial and error was needed, so the suggestion offered by Dempster (*24*) was employed. Dempster suggested that an analysis of longitudinal changes in functioning might reveal clusters of variables which changed together; these would show subsystems which aged together. A factor analysis of change scores was therefore attempted to identify major subsystems which showed consistent patterns of change.

Factor analysis of change scores.—If aging is a single process, as implied by the notion of functional age, then a factor analysis of change scores from one time to another should show a single general factor, "rate of aging," together perhaps with specific factors for specific variables. If rates of aging are constant in individuals, then change from time one to time two should correlate positively with change from time two to time three, three to four, and so on. If major subsystems which age at different rates exist, they should emerge as distinct factors. If none of these is true, if changes in one variable are unrelated to changes in other variables, or to changes in the same variable over other time periods, then the whole idea of "an" aging process becomes suspect.

These ideas were examined by an exploratory factor analysis of change scores over two successive 5-year periods for a variety of biomedical, anthropometric, and psychosocial variables. Forty-seven variables in all were entered into the principal components factor analysis, and all factors with eigenvalues greater than 1 were retained for Varimax rotation.

Before proceeding to the results, it is necessary to note that there is a technical flaw in the analysis. Because of regression to the mean, raw change scores are poor indicators of real change. Ideally, residual gain scores would have been used instead, but that was technically infeasible at the time. To some extent, therefore, the analysis contains error, and results must be viewed with caution.

Twenty factors had eigenvalues greater than 1, and the largest was 2.58. Table I–4 lists the prominent loadings for the 20 factors. The results are quite clear: over a 10-year interval, there is no general factor of rate of aging, nor any major subsystems showing specific rates of aging, nor any consistency between one time period and the next, except for that artifically introduced by error or instability of measurement. Change in systolic blood pressure at interval one, for example, is negatively related to change over the second interval, because blood pressure is so variable in the individual that high measurements occur mostly by chance, and are therefore mostly succeeded by relatively lower measurements. This artifact accounts for most correlations above 0.20.

However, aside from this artifactual difficulty, there is another serious objection, namely, that a 10-year interval is insufficient time in which to detect processes of aging. Everitt (*2*) has shown that even the most strongly age-related variables in the biomedical domain rarely change more than 50 percent over the course of the adult life span. If that interval is taken to be 50 years, a 10-year span will mean a change in only 10 percent at the most. This figure is consistent with the percent changes listed in table I–5.

Cross-sectional measurements on individuals are in this respect more sensitive than longitudinal research in terms of detecting aging differences, because cross-sectional research draws on the cumulative

Table I–4.

Variables loading 20 varimax-rotated factors of change scores for two 3-year periods

Factor			Eigenvalues of correlation matrix (percentage)
Factor 1:			
	T_2–T_1	Systolic 0.77	2.58
	T_2–T_1	Diastolic 0.68	(5.5)
	T_3–T_2	Systolic −0.73	—
	T_3–T_2	Diastolic −0.73	—
Factor 2:			
	T_2–T_1	Bideltoid 0.80	2.29
	T_2–T_1	Abdominal depth 0.82	(4.9)
	T_2–T_1	Triceps 0.70	—
Factor 3:			
	T_3–T_2	FEV$_1$ 0.84	2.16
	T_3–T_2	PVC 0.83	(4.6)
	T_2–T_1	FEV$_1$ −0.20	—
	T_2–T_1	PVC −0.23	—
Factor 4:			
	T_3–T_2	Earlength 0.77	2.01
	T_3–T_2	Ear breadth 0.84	(4.3)
	T_2–T_1	Ear breadth −0.38	—
Factor 5:			
	T_2–T_1	Hemoglobin 0.84	1.92
	T_3–T_2	Hemoglobin −0.81	(4.1)
Factor 6:			
	T_3–T_2	Bideltoid −0.70	1.88
	T_3–T_2	Abdominal 0.81	(4.0)
	T_3–T_2	Triceps 0.67	—
Factor 7:			
	T_2–T_1	Grip Strength 0.83	1.78
	T_3–T_2	Grip Strength −0.85	(3.8)
Factor 8:			
	T_2–T_1	CMI A–L 0.76	1.61
	T_2–T_1	CMI M–R 0.85	(3.4)
Factor 9:			
	T_2–T_1	Electrolyte Sed. Rate 0.82	1.61
	T_3–T_2	Electrolyte Sed. Rate −0.81	(3.3)

Factors			[illegible]
Factor 10:			
	T_2-T_1	PBI −0.88	[illegible]
	T_2-T_1	PBI 0.82	([illegible])
Factor 11:			
	T_2-T_1	Greyness of hair 0.85	[illegible]
	T_2-T_1	Greyness of hair −0.75	([illegible])
Factor 12:			
	T_2-T_1	Glucose −0.90	[illegible]
	T_2-T_1	Glucose 0.83	([illegible])
Factor 13:			
	T_2-T_1	Cholesterol 0.87	[illegible]
	T_2-T_1	Cholesterol −0.80	([illegible])
Factor 14:			
	T_2-T_1	FEV_1 0.78	[illegible]
	T_2-T_1	FVC 0.78	([illegible])
	T_2-T_1	BUN −0.38	—
Factor 15:			
	T_2-T_1	Baldness 0.90	[illegible]
	T_2-T_1	Baldness −0.76	([illegible])
Factor 16:			
	T_2-T_1	Bone Conduction 4kHz 0.76	[illegible]
	T_2-T_1	Speech Reception Threshold 0.71	([illegible])
Factor 17:			
	T_2-T_1	Sitting Height 0.71	[illegible]
	T_2-T_1	16PF Tendermindedness 0.53	([illegible])
	T_2-T_1	Ear breadth −0.44	—
Factor 18:			
	T_2-T_1	CMI A-L 0.82	[illegible]
	T_2-T_1	CMI M-R 0.71	([illegible])
Factor 19:			
	T_2-T_1	Earlength 0.76	[illegible]
	T_2-T_1	Sitting Height 0.53	([illegible])
	T_2-T_1	16PF Surgency −0.38	—
Factor 20:			
	T_2-T_1	Nose breadth 0.80	[illegible]
	T_2-T_1	Nose breadth −0.45	([illegible])

effects of a lifetime of aging. Appealing as the idea of predicting longitudinal changes is, it may be that the fairest test of functional age would use status at one time as the criterion rather than change over so short an interval as 10 years.

Predicting functional status at one point in time.—A series of multiple regressions were conducted resembling those used to predict changes in

Table 3-3.
Percent changes over 5- and 10-year intervals for age-related variables

Variable	Percent change		
	T_1-T_2	T_2-T_3	T_1-T_3
Baldness	[illegible]	[illegible]	[illegible]
Grayness	[illegible]	[illegible]	[illegible]
Ear length	[illegible]	[illegible]	[illegible]
Air conduction, 8 kc	[illegible]	—	—
Skinfold	[illegible]	[illegible]	[illegible]
Abdominal depth	[illegible]	[illegible]	[illegible]
Triceps	[illegible]	[illegible]	[illegible]
Sitting height	[illegible]	[illegible]	[illegible]
Nose breadth	[illegible]	[illegible]	[illegible]
Ear breadth	[illegible]	[illegible]	[illegible]
Grip strength	[illegible]	—	—
Cholesterol	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]
Glucose	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]
Hemoglobin	[illegible]	[illegible]	[illegible]
[illegible]	—	[illegible]	—
FEV	[illegible]	[illegible]	[illegible]
FVC	[illegible]	[illegible]	[illegible]
Systolic blood pressure	[illegible]	[illegible]	[illegible]
Diastolic blood pressure	[illegible]	[illegible]	[illegible]

functioning, but predicting instead simple level at the second cycle of measurement. The same set of variables—grayness, ear length, speech reception threshold, dynamometer, and lead matching—were entered on the first step of the regression; chronological age was again entered on the second. The logic behind this analysis was as follows: If some combination of functional age predictors really represents the point on the life-course of the individual, then it should be more closely related to performance on age-related variables than simple chronological age is. Note that this is the most straightforward test of the promises that the advocates of functional age make. They claim that measuring the functioning of the individual on a series of appropriate measures will be a better index of his overall ability to function than chronological age is; if so, they should yield a higher correlation with measures of biological and psychological functioning.

Table 3-4 shows the multiple R for the five functional age predictors, the simple r for chronological age, and the combined multiple R for all six. While the absolute value of the correlation is much higher here than it was in the attempt to predict changes, the basic pattern is the same: simple chronological age is as good or better than the best combination of functional age predictors in almost all cases. In predicting a few of the anthropometric variables some incremental validity is shown by the functional age predictors, but it is on these marginal and meager results that the hopes of functional age hang.

Table [illegible].
Regression predicting level of age-related variables from functional age predictors and chronological age

Variable	df	Multiple R²a	Simple rb	Total multiple Rc
MEFR	[illegible]	[illegible]	[illegible]	[illegible]
FEV	[illegible]	[illegible]	[illegible]	[illegible]
Systolic blood pressure	[illegible]	[illegible]	[illegible]	[illegible]
Cholesterol	[illegible]	[illegible]	[illegible]	[illegible]
Glucose	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
Abdominal depth	[illegible]	[illegible]	[illegible]	[illegible]
Triceps	[illegible]	[illegible]	[illegible]	[illegible]
Sitting height	[illegible]	[illegible]	[illegible]	[illegible]
Grip strength	[illegible]	[illegible]	[illegible]	[illegible]
GATB "Intelligence"	[illegible]	[illegible]	[illegible]	[illegible]

* p<.05 ** p<.01 *** p<.001

a With functional age predictors: [illegible]

b With chronological age

c With chronological age and functional age predictors

To be sure, these results are not definitive—scientific results never are. The Normative Aging Study population was selected for health, and this may obscure the effects of aging, although it has the advantage of unconfounding the effects of aging and disease, at least to some extent. So long as there is a perceived need for functional age, there will be a tendency to try to salvage it, regardless of the evidence. The last task, therefore, of one who would dispose of the concept is to provide alternatives to it. A few suggestions are offered in the summary and conclusions below.

Conclusions and Recommendations for Future Research

In summary, it may be said that the idea that a set of functional indicators might provide a better estimate than simple chronological age of the point on the life-course that an individual had attained led to several attempts to generate a formula for functional age. While this notion has considerable intuitive appeal, on closer examination it disregards much of what is known about the aging process and about the sources of variance in measures of specific biological and psychosocial variables. Regressions pitting functional age predictors against chronological age in general point to one conclusion: that the best indicator of the aging process, or rather, of the aging processes, is provided by calendar time.

The explanation for this phenomenon is not hard to find: chronological age, as a measure of the universal medium of events, is more or less closely related to the whole multitude of causal sequences which contribute to the overall process of aging. If a single mechanism of aging were responsible for all the characteristic changes seen with age, then the concept of functional age would probably prove useful. But aging results from a variety of normally independent sequences of events: the accumulation of trauma, the random decay of genetic transmission, genetically programmed functional changes, disease processes, and all the complex interactions of these factors in various systems of the body. All these processes share in common only one thing: they take time. And the best measure of the time they take is chronological age.

Chronological age has in addition a number of practical advantages over any form of functional age. Few variables can match its reliability or day-to-day stability, or the sheer convenience of asking the individual for his or her age. Where the possibility of error or deliberate falsification is suspected, our society provides a series of public records (birth certificates, etc.) which can be used to check. And chronological age is a universal variable. Greyness cannot be assessed in the totally bald nor hearing loss in the congenitally deaf, and baldness is rarely a useful index of aging among women. But chronological age is completely democratic, possessed equally by rich and poor, man and woman, healthy and sick. Some combination of all these factors may help explain why we call the processes of decrement which lead ultimately to death—aging.

McFarland and [illegible] (7, 8) argue cogently that the use of an arbitrary age cutoff for mandatory retirement is inequitable, since ability to perform satisfactorily in a job is only crudely indexed by age. The attempt to remedy this situation by substituting for chronological age some measure of functional age, however, is no improvement. If it is arbitrary to retire a 65-year-old man simply because he is 65, how much more so to retire a 50-year-old who happens to be prematurely grey? Given the tradition in this culture of reliance on chronological age, the validity of functional age would have to be demonstrated beyond a reasonable doubt before it could become the basis of a social policy that would not only be more equitable, but would also seem more equitable to the members of the society.

And in any case, there are much more direct criteria for retirement. The age of retirement should be fixed by the ability to perform competently in the job. A judge should be retired when he becomes senile, but not when he loses manual dexterity. Every category of job should develop its own appropriate and valid criteria of performance, and hire, fire, and retire on that basis. Until such criteria are established, the established criterion of chronological age remains the best approximation.

The tradition of longevity research is a long and well-established one and has proven its worth both as a tool for practical, actuarial purposes and for investigating causal influences in the aging process. Like chronological age, death is a universal, reliable, and stable criterion, and is not likely to be replaced in research. The attempt to anticipate the results of longevity research by gauging the functional age of living individuals does not look promising, nor is it necessary in order to do research on topics such as environmental impacts. If, for example, it is hypothesized that pollution will adversely affect liver function, this can be directly tested by measuring liver function in individuals living under varying conditions of pollution. The alternative of measuring some hypothetical "functional age" and attempting to infer from a difference in mean functional ages of individuals something about the aging of their livers is cumbersome and silly.

If there were a single process of aging, if environmental impacts had uniform effects on all organs and systems of the body, then a single measure of functional age could be profitably used. But all evidence flies in the face of these assumptions. The alternative of investigating singly all the biological and psychological processes is more time consuming and less dramatic, but it is a course dictated by the nature of the phenomena of aging.

In general, the independence of aging processes seen in the analyses presented here, and in the well-known different curves of decline with age for different functions, leads to the inevitable conclusion that research must for the time being remain specific. Chronological age is indeed a dummy variable, and research should attempt to replace it in every case with an account of the real etiology of age-related changes. The substitution of a new dummy variable, functional age, will not be a step forward on this process; indeed, it would be a step back.

It should not be assumed from what is said that there are no relations between different systems in the aging process. Deterioration of the cardiovascular system may lead to senility, and that will have profound effects on social behavior and status. But such interactions are complex phenomena which may occur only in the extreme ends of the aging process, or only in actual disease conditions. The changes which occur with time do not march arm-in-arm toward death to the drumming of "functional age," but rather execute a more subtle and complicated dance which only painstaking and systematic research will uncover.

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Discussion

Palmore: If you had in your criteria some measure of remaining longevity, would you also find that functional age is no better a predictor than chronological age?

Costa: It is difficult to say. I have only been able to evaluate a very small sample—about 42 deaths. We did not find any age-related predictors for longevity but we did find very specific pathogenic events which occurred more in older subjects but were different from such factors as greyness of hair, speech reception and the like. With a larger sample, it might be different.

I felt at first that, if the concept had any utility, it might be with an appropriate criterion like longevity. I feel, though, that if you have a set of indicators that is perfectly correlated with age and is a perfectly reliable alternative to chronological age, it is also perfectly useless.

Adelman: It is reasonably clear that one can find functional age changes. Biologists can do it in well-defined experimental populations, something which epidemiologists may not be able to define through clinical studies. Certainly, there are parameters which do change as a function of age and which do relate to species of different life spans. The difficulty is being able to define what you are dealing with and recognizing that with something as complicated as a human population, different functions are going to deteriorate at different rates and at different times in different people.

Greenhouse: Dr. Costa's point, as I gather, is somewhat subtle. He is not arguing against the existence of factors which could be thought of as functionally changing in an aging person or that there are no functional factors or organ systems which change with age; he is saying that when you try to put together the research findings in an attempt to identify any such system, it does not turn out to predict one thing or another. The comment has been made that he should limit his conclusion to those factors which he studied in his analysis. Then greyness and baldness may not be the characteristics of any system which would represent a functional age characterization. I do feel, however, that the onus is on the other side. He is not saying that you will not find other systems which change with age; he is saying that you will have to demonstrate that functional age or anything else predicts better than the system which he now has.

Kasl: Dr. Costa, you determined the components of functional age by seeing how they correlate with chronological age. What is the justification for the rationale underlying the procedure for developing components of functional age? Since functional age is supposed to be something different conceptually from chronological age, why do you look to see how they are correlated?

Costa: You are mentioning the point which the paper identified as the major stumbling block in the use of functional age. I have been a vigorous opponent of the operational use of chronological age as a criterion of functional age in a regression model.

Kasl: You are a psychologist and are familiar with the literature on masculine and feminine characteristics in which anything that differentiates males from females is thrown into the study. This sort of thing lacks a conceptual basis.

Costa: I presented what the advocates of functional age have done. As recently as 1976, in the *Journal of Gerontology*, there are reports attempting to assess biological age or functional age by predicting chronological age from sedimentation rates, changes in pulmonary function, and the like. Certainly I agree that the model and its typical operational form is lacking in any sound conceptual basis.

Kasl: There seems to be no conceptual input at all.

Costa: Yes, but to give it the benefit of the doubt, I wanted to see whether anthropometric indicators among others would indeed tap a differential rate of aging. I found they did not.

Siegel: It seems to me that we must distinguish between a concept of aging that is applicable to individuals and a definition or measurement device which would apply to groups when we are talking about the implementation of social programs. Nonchronological age may be useful for individual analysis, but for groups chronological age is essential as a basis for the administration of public programs, such as retirement programs.

Suppose you had two groups of people, both in the age group 60 to 64 but each group having different "aging" characteristics. Would you say that their aging cannot be distinguished simply on the ground that chronological age is the sole and best way of measuring their aging?

Kovar: There must be many measures of functional age. If one does not work, we should not stop searching for others which might be more effective. In other words, there must be other measures of functional age besides greyness which may be more effective; we must look for it.

Costa: I want to say that I did not have the time to spell out the various meanings of this concept. I do not want to appear to be arguing that we should not look at age-related changes in specific functions. But that is only our starting point; the main point is to identify the causal mechanism underlying the changes.

As a basis for social policy, the concept has real meaning. For example, advocates of the use of functional age—epidemiologists, social scientists, behavioral scientists—want to replace the arbitrariness of retirement of persons at age 65. My point is that using functional age is no improvement; in fact, it might be a step backward to argue that you should retire a judge or any other person for greyness or hearing loss.

Weksler: It is possible that chronological age might be the best definition of aging from birth to death; however, at any time segment or any age within 1 year, or 5 years, or 10, certain functional aspects may be more powerful. Is it possible that the limitation you found is that nothing is better than chronological age from birth to death but that within certain segments, the predictors of life span or longevity at age 60 might be A, B, and C, which would be better than chronological age? On the other hand, these predictors might not be better at age 50 or 70.

Greenhouse: To clarify Dr. Siegel's point further, let us assume we have two 67-year-olds, both of whom are retired. One is still actively playing tennis and consults for the NIH, etc., and the other is debilitated, living in a nursing home and cannot ambulate. Chronological age cannot distinguish these two individuals.

Atchley: Many people have tried the idea of functional age in a number of different ways. The conclusion, whether sociological, biological or psychological, is that composite indicators are not very feasible.

Costa: The alternative pursuit of investigating each of the biological and psychological processes that change in an orderly fashion with age is more time-consuming and less dramatic than a composite search for functional age. It is, however, dictated by the nature of the phenomena of aging.

Shanas: We all realize that in studies of human populations we are dealing with populations in different environments. Not only must we include in our sample those who are free of atherosclerotic changes or at the same level of change, we have to isolate the subgroups of the population that are living in somewhat comparable environments. We are talking about functional age which in itself is a confounding variable and which we do not yet know how to operationalize.

Costa: That is correct. Some literature indicate that precipitous changes in intellectual functioning are more useful as predictors for longevity than chronological age.

Weksler: One would therefore suggest against the chronological age index; one should develop functional assessment capabilities. We would then have a tool from 30 to 40, 40 to 50, 50 to 60, which would be more powerful as predictors than chronological age.

Costa: I think the concept of functional age obscures more than it clarifies. Certainly more precise measures of systems functioning at each calendar year might be useful. But are they then really replacing chronological age?

Closing Remarks by Chairperson Samuel W. Greenhouse, Ph. D.

Any attempt to summarize these four excellent papers in a brief, cursory manner would do their authors an injustice. Instead, may I just note those remarks which bear on possible future research programs in epidemiology.

Dr. Butler points out that in studying the aging process in humans, one is faced with a complex phenomenon involving the action and interaction of multiple factors in both antecedents and outcomes. He therefore concludes that increased understanding of aging requires a multidisciplinary approach and that epidemiology will have to explore interactive processes and be concerned with analytic procedures.

Dr. Adelman asks what is the most appropriate component in a definition of biological aging that may facilitate epidemiologic studies. He suggests this component to be one which characterizes all aging populations, namely, the "progressively modified ability to adapt to changes in the surrounding environment." Thus, the epidemiologist "should think in terms of which biological adaptations are most relevant to the question at hand."

Although Drs. Weksler and Costa do not mention epidemiology specifically, what they discussed has significant implications for the epidemiologist in aging. Dr. Weksler's thesis, in effect another component in the definition of biological aging, is that the genetic control of life span is mediated through two biological parameters: the proliferative capability of somatic cells and normal immunologic response. I suppose this means that major epidemiologic studies should incorporate some measurements of these biologic functions. But these may be easier said than able to be put into practice. Epidemiologists deal with hundreds of study subjects and it is not clear whether drawing bloods and rather complex laboratory workups are feasible. Another difficulty is whether agreement can be reached by geneticists and immunologists on the choice of a small number of characteristics to be measured that are effective estimates of the biological parameters.

Dr. Costa's paper and the ensuing discussion would seem to deprive the epidemiologist of one of his surest variables, age. This applies also to the epidemiologist who is not just interested in aging. Age has been used as an objective variable for control or stratification purposes. In studies of aging, chronological age will probably still be used as a global measurement. Given what we have learned from this morning's discussion, the concept of functional age may still be in an experimental phase, at least as far as its use as a general measure of age is concerned. In some studies, it evidently may be possible to use specialized definitions of functional age, but as a supplement to chronological age and not as its replacement.

One thing is clear. This meeting is only the beginning of a series of discussions which ought to result in more effective research programs in the epidemiology of aging.

Session II

Biomedical Correlates of Aging

Opening Remarks by Chairperson George A. Sacher

This session is concerned with the biological correlates of aging. Before we begin, I would like to make one remark which I think will have some orienting value. In my work, which is confined to animal populations, the definition of aging that I have found to be extremely useful is an actuarial definition: aging is the pattern or course of mortality as a function of age. More explicitly, the age-specific mortality rate—how it varies with age, and what happens to it when we introduce some sort of environmental perturbation, which might either be toxic or beneficial.

Figure II–1 shows the recalculated results of experiments (*1*, *2*) in which caloric restriction is introduced. On the ordinate, we have the age-specific death rate, with the solid symbols and the X's being the control populations, and the open symbols the restricted populations. We see that in the calorically restricted populations the slope of the line which is the rate at which the logarithm of the mortality rate increases with age, is decreased compared to the control. The implication here is that the process of aging has somehow been slowed down. However, if one looks at any of the life-extension experiments in which various pharmacological agents have been employed, one observes a different outcome. Figure II–2 shows the recalculated results of an experiment (*3*) in which procaine was administered (a proprietary novocaine preparation) to rats, periodically throughout life. There is a decrease in the death rate at every age in the treated population, which is not progressive. It is a constant, parallel displacement, which says that there is a beneficial effect on the population, but it is not a cumulative kind of effect, such as seen in figure II–1.

Other chemical agents that have been tested for life-prolonging efficacy include antioxidants, catecholamine precursors, membrane-stabilizing agents and steroid hormones. Insofar as any effect on the life table was observed, in every case it was of the form seen in figure II–2, i.e., a noncumulative decrease of mortality rate by a constant factor. However, all of these were tested as "anti-aging" treatments, and the increased survival observed was attributed to a reduction of the aging rate. What was observed was a protective action, but it did not reduce the aging rate. It is evident that a more penetrating analysis of the biological basis of life table modification is needed (*4*).

Figure II–3 represents the two kinds of outcomes in schematic form. If we introduce any kind of life table modification treatment, and maintain it throughout adult life, the natural progression of aging can be modified either with a reduction in the rate of aging (the C curve) or in a reduction of the intercept parameter that I call the initial vulnerability (B curve) (*5*).

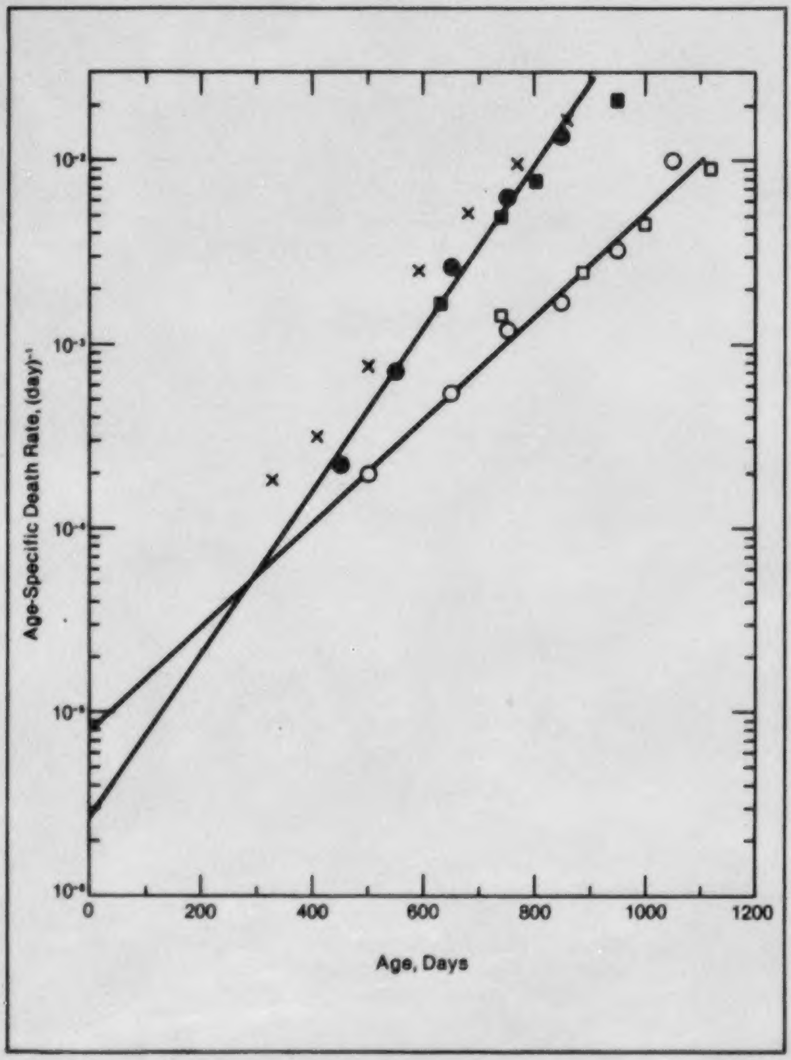

Figure II-1.
Effect of caloric restriction on the age-specific death rate of rats. (References 1, 2, 4.) Solid symbols and X are controls; open symbols are calorically restricted.

The essential point here is that from the standpoint of an actuarial measure of aging, which is essentially a population measure, there are at least two parameters to consider in the adult population. These two parameters can differ because of genetic factors, so it follows that there

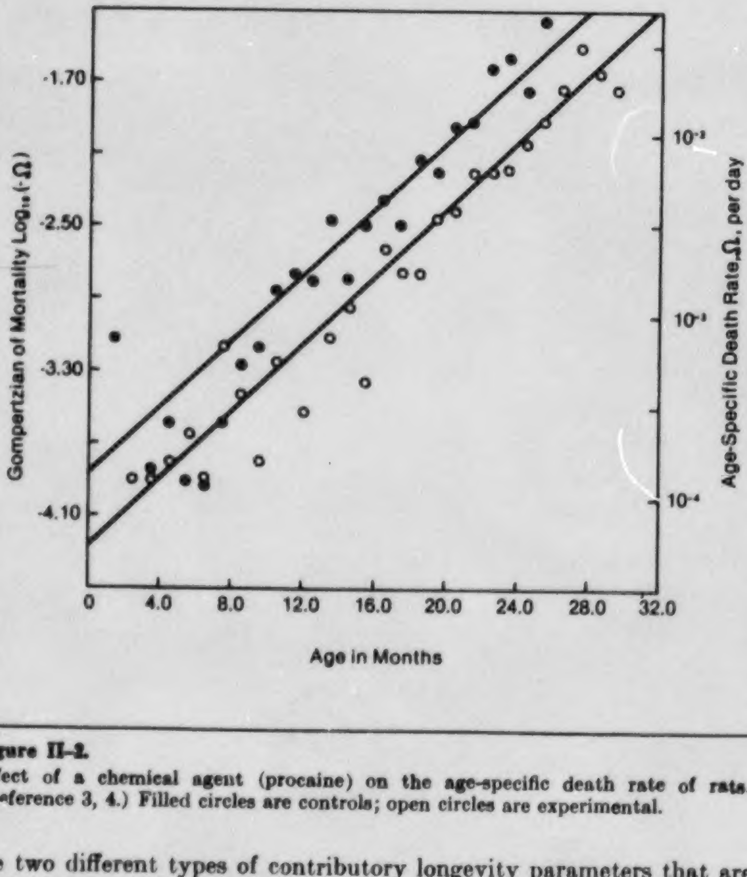

Figure II-2.
Effect of a chemical agent (procaine) on the age-specific death rate of rats. (Reference 3, 4.) Filled circles are controls; open circles are experimental.

are two different types of contributory longevity parameters that are under independent genetic control. There is little direct evidence about how these two parameters are distributed in human populations, but I offer the conjecture that any variance of longevity in human populations is due primarily to variance of the vulnerability (intercept) parameter and not to the rate-of-aging. (slope) parameter. There is some support for this in animal studies and in aging and disease epidemiology in human populations (*4*).

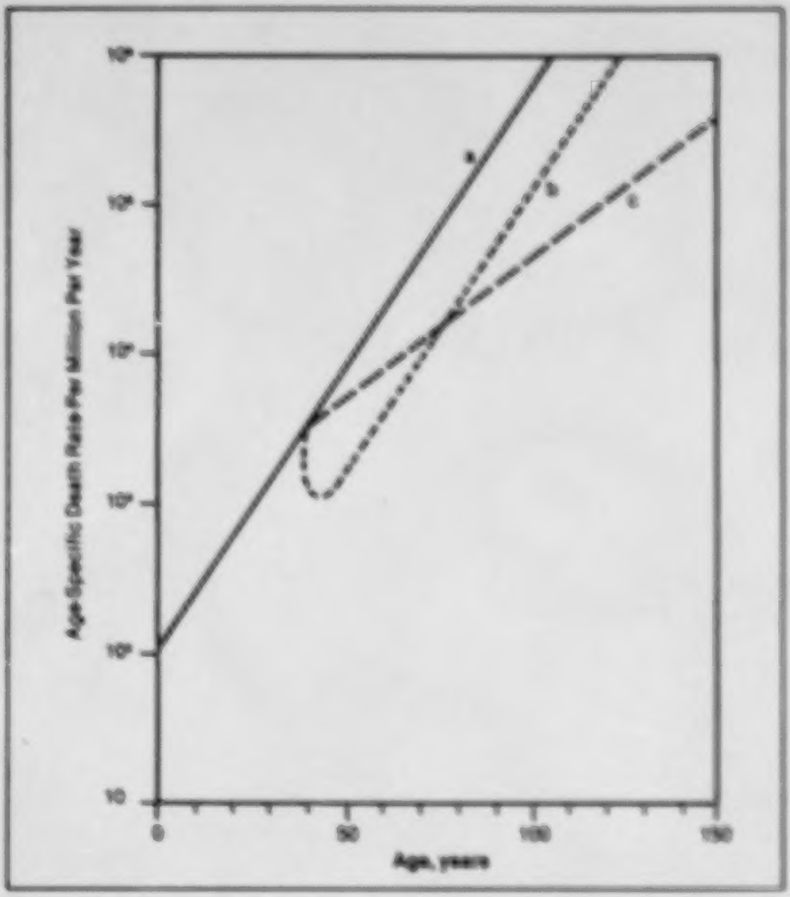

Figure II-3.
Schematic representation of life table of modification treatment outcomes.

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Predictors of Longevity

Erdman Palmore, Ph. D.

Center for the Study of Aging and Human Development
Duke University Medical Center
Durham, NC 27710

A recent cartoon shows a very old man telling another old man the secret of his longevity: "The secret of my longevity is a balanced diet, adequate rest, and plenty of money." This illustrates the two main types of predictors of longevity: behavioral factors which the older person may be able to control such as diet, rest and exercise, smoking, work or retirement, and social activity; in contrast to those characteristics which the older person cannot control such as heredity, sex, race, intelligence, and socioeconomic status.

In this paper I will briefly review the research on longevity and our research at Duke to summarize what has been found and some of the questions that these findings raise (*1*). I will conclude with a discussion of some study designs that may be able to answer some of these questions.

Behavioral Predictors of Longevity

The first group of predictors of longevity include the behavioral factors which lend themselves to control by the individual. These include:

1. *Diet.*—The main thing we know about diet is that too much or too little food reduces longevity. For example, in the First Longitudinal Study at Duke, we found that a tenth of our participants with the lowest weight/height^2 ratio and the tenth with the highest weight/height^2 ratio both had substantially more illness and less longevity than those in the middle 80 percent (*2*). This is such a well-known relationship that obese persons must pay higher life insurance premiums to compensate for their greater mortality risk. However, since obese persons usually get little exercise, there remains a question as to how much of their increased mortality may be caused by their lack of exercise. As far as I know, no one has satisfactorily separated the effects of obesity and lack of exercise.

High cholesterol in the diet has also been found to be associated with higher mortality, especially from cardiovascular diseases. However, this relationship may not hold for persons who survive to old age. In our first longitudinal study, we found a small positive association between serum cholesterol levels and longevity. This finding needs testing on other samples of older people.

2. *Exercise.*—Several studies have found that older persons who are more active and get more exercise tend to live longer than those with less exercise. For example, we found a significant positive relationship between the number of leisure activities engaged in and longev-

ity. But in addition to the problem mentioned above, that lack of exercise tends to accompany obesity, there is the possibility that greater exercise may just be an indicator of better general health which causes the greater longevity. The resolution of this question may require controlled environmental studies.

3. *Smoking*.—The association between cigarette smoking and greater mortality is now well established. The question of whether pipe smoking is also associated with significantly greater mortality is still controversial. The U.S. studies found little or no greater risk among pipe smokers, but recent studies in Europe (where there may be more inhaling among pipe smokers) indicate substantially greater risks. However, it is possible that the greater longevity of nonsmokers may be partly due to their being more careful about their health in general, or may be due to their tending to be of a different personality type (such as type B) which increases their longevity. Even the findings which show that persons who gave up smoking have greater longevity than those who continue to smoke, may be due to a selective process in which the more health conscious or otherwise healthy persons were willing and able to stop smoking.

4. *Retirement and work*.—There is no question that retired persons have higher mortality than those who continue to be gainfully employed. But this may be entirely due to the selective factor that only the more healthy aged continue to work while most of the sicker persons retire. Studies of persons in compulsory retirement systems (where both the healthy and the sick must retire) find no significant difference between expected and actual mortality rates in retirement. Yet, we found that a scale of "work satisfaction" was one of the strongest predictors of longevity in the Duke studies. This scale included satisfaction with housework, hobbies, and voluntary activities as "work" and thus may be more a measure of satisfaction with a meaningful role, rather than with employment only. It may be that forced retirement from a satisfying job which is not replaced by some other satisfying role leads to greater mortality, but that a desired retirement from a stressful job may lead to greater longevity. Thus, we need to find out under what conditions retirement leads to greater mortality or greater longevity.

5. *Marital status*.—We know that among the aged, married persons have lower mortality rates than any of the nonmarried categories. But several puzzles remain. Never married males have higher mortality rates than widowed males; but among the females the widowed have higher rates than the never married. Does this mean that for older women, it is better to have never married than to be widowed? What is the true explanation of the higher mortality among the widowed compared to married (for both men and women)? Is it the shock and loneliness of widowhood that increases mortality, or is it that whatever life-style and environmental influences were related to the death of the spouse tend to hasten the death of the widowed person? We have found little or no evidence of lasting trauma caused by widowhood in our longitudinal studies (9). Indeed, many found the death of a long ill spouse a kind of relief.

6. *Social activity.*—The Duke studies found that various measures of greater social activity were modestly correlated with greater longevity. Similarly, the National Institute of Mental Health (NIMH) longitudinal study of 47 very healthy men found that one of the best predictors of their longevity was a rating of the number of activities and organization of their activities. It seems plausible that persons with more social activities will better maintain their physical and mental functioning, as well as feelings of self-esteem, belonging, and social support, all of which may contribute to greater longevity. However, we again have the chicken and egg problem: is it greater social activity that leads to better health and longevity, or better health that leads to greater social activity?

Less Modifiable Predictors of Longevity

In contrast to the behavioral factors, we have those characteristics which may seem of less practical interest since, by definition, they are those which the individual cannot change (or is unlikely to change) in old age. However, in addition to their theoretical interest, they may have implications for social policies and programs which may increase those aspects of the characteristics which contribute to the greater longevity. These include:

1. *Heredity.*—Many studies have shown a general association between longevity of parents and of offspring. However, we do not know how much of this association, if any, is due to genetic heredity and how much is due to the inheritance of social, psychological, and economic environments similar to those of their parents. Whatever environmental and life-style influences contributed to the longevity of the parents are likely to be passed on in the family and contribute to the longevity of the offspring. Of course we know some diseases are genetically inherited, such as sickle cell anemia, but these tend to take their toll early in life and not affect the longevity of those who survive to old age. In fact, our studies of persons over 60 found almost a zero correlation between age of parents at death and the longevity of their aged offspring. In our study, apparently whatever genetic influences on mortality there may be earlier in life, these tend to be washed out by a lifetime of environmental influences; so that among those healthy enough to survive to age 60 and enter our study, there is no longer any discernible genetic influence on their remaining longevity.

This has important practical implications, because if longevity among the aged were mainly controlled by one's genes, there would be little point in trying to do anything about longevity. On the contrary, longevity among the aged appears to be mostly the result of environmental and life-style differences, many of which can be changed.

2. *Sex.*—Another paper in this conference deals with differences in longevity between men and women, so I will simply raise the question as to how much of this difference is due to sex-linked genetic differences and how much is due to differences in life-style of men and women. Based on the ratio of male to female mortality rates at various ages, I

would estimate that about half the greater longevity of women is due to genetic differences and about half is due to differences in life-style, such as less hazardous occupations and more careful driving which produce lower accident rates, less cigarette smoking which produces less lung cancer and cardiovascular disease, etc. We need more studies to find out how much the sex difference in mortality rates can be reduced by controlling for such variables. According to the theory of life-style difference, as women become more "liberated" and similar to men in their life-styles, we should see a convergence of male and female mortality rates.

3. *Race.*—It is well known that blacks have greater mortality at most ages than whites; and that most or all of these differences are probably due to the differences in socioeconomic status and life-style. However, beyond age 75, the statistics seem to show that blacks have *lower* mortality than whites. One theory to explain this switch around age 75 is that only the hardier blacks survive to such an age because of their more difficult environments and this "selection of the fittest" accounts for their lower mortality rates. I tend to believe the other explanation: that this is due to a greater exaggeration of age among blacks because of less documentation of their true date of birth. A study is needed that would compare mortality rates among only those blacks and whites with documented birth dates.

4. *Intelligence.*—Several studies have found that higher intelligence or better mental functioning is associated with greater longevity. But there are several alternative explanations of this relationship. Better mental functioning in old age could be just an indicator of better general health which would produce greater longevity. Or the association between intelligence and longevity could be explained by the association of intelligence with higher socioeconomic status, which is the real cause of greater longevity. Or it may be that higher intelligence allows a person to solve problems and cope with crises better so as to increase longevity. When we have controlled for other variables in our study, intelligence was no longer a statistically significant predictor of longevity. This suggests that it is not intelligence itself that contributes to longevity, but the other variables with which intelligence is associated.

5. *Socioeconomic status (SES).*—Several studies of the adult male population have found that there are marked differences between upper and lower occupational and educational groups. For example, in the United States white males ages 25 to 64 who were professional or technical workers had mortality rates 20 percent below average, while those who were laborers or service workers had mortality rates 37 percent above average (*4*). An exception to this general white-collar/blue-collar difference is the fact that agricultural workers had the lowest mortality of all occupations: 24 percent below average. Apparently farming in the United States is generally a healthy occupation. Despite this exception, education and income are also strong predictors of longevity, which supports the cartoon's claim that "plenty of money" can be a secret of longevity.

However, there have been few studies of SES and longevity among the aged. In a followup study of all persons over age 60 living in Chapel Hill, North Carolina, we found the same general relationship between SES indicators and longevity (5). But, in contrast to the national studies of persons aged 25 to 64, we found that farmers had the *lowest* longevity of any group. This difference between the national studies and the Chapel Hill study could be due to the characteristics of farming in this region, or it could be due to the difference in age groups. Perhaps farming has moved from a relatively unhealthy occupation to a relatively healthy one in the last couple of generations. Replications of our study in other regions are needed to answer this question.

But given the generally positive relationship between SES and longevity, there are two major alternate explanations: "nature versus nurture." The "nature" or hereditary theory attributes the differences in longevity to genetic differences between SES levels. This theory argues that longevity is largely determined by one's genetic constitution and the reason that higher SES categories have greater longevity is that genetically superior individuals tend to move up to, or remain in, the upper SES categories; while generally inferior individuals tend to move down to, or remain in, the lower categories.

The "nurture" or environment theory attributes the differences in longevity to the environmental differences between SES levels. It argues that the greater longevity of the upper SES levels is due to their superior diet, housing, medical care, education, and less hazardous occupations and life-style. Probably both theories are partially true and the only problem is determining the relative importance of nature and nurture for specific groups. Studies of twins that have been separated at birth are a theoretically promising way to study this problem, but so far these studies are inconclusive because of their small numbers and because the separated twins are usually placed in similar SES families.

This problem has important practical and policy implications, because if the "nature" explanation is the major explanation, there is little that improving SES levels can do to increase longevity. On the other hand, if the "nurture" explanation is the major one, then eliminating poverty, improving diet, housing, medical care, etc. could substantially increase longevity.

Promising Designs and Summary

To measure the importance of predictors other than age and sex on longevity, we have developed a tool for standardizing longevity which we call the longevity quotient (LQ). In other words, just as the intelligence quotient (IQ) standardizes for the effects of age on intelligence, by dividing the actual test score by the expected score for a person's age group; so the LQ standardizes for the effects of age and sex on longevity by dividing the *actual* years an individual survived from the beginning of a study by the *expected* years based on actuarial tables of life expectancy by age and sex groups. This, then, gives each individual a standardized longevity score and multiple regression techniques can be used to control for various factors and estimate their separate and joint effects on longevity, and to develop a prediction equation which takes all measured variables into account simultaneously. Studying group mortality rates usually makes such a multivariate approach impossible because the cells quickly become so small that the rates become unreliable.

Large-scale longitudinal studies of the aged may be the ideal method (short of experimentation) to develop better predictors of mortality. In such studies specific age cohorts are followed over a substantial number of years (say 10 or more), so that information gathered at the beginning of the study can be used to predict mortality and longevity for various types of persons. The major disadvantages of such studies are the long time they take, the relatively large cost, and the problem of keeping track of participants who move. However, several government agencies such as the Social Security Administration, the Department of Labor, and the Office of Economic Opportunity are now conducting large scale longitudinal studies, and the analysis of factors predicting longevity in these cohorts could be added to their main focus at little additional cost.

In order to avoid the time and costs required by longitudinal studies, various kinds of retrospective designs utilizing records and past surveys might be developed. For example, one might study a cohort of veterans of World War I using Army and Veterans Administration records to correlate their longevity with their scores on their intelligence tests, health examinations, subsequent occupations, etc.

Another retrospective design would be to take a sample of persons who have achieved great longevity (say 80 years or older) and compare their characteristics at an earlier point in time (say 30 years ago) with those of their age mates, based on the U.S. Census or other representative surveys. The characteristics that differentiated the surviving 80-year-olds from the rest of their age cohorts (most of whom would be dead) could be predictors of longevity.

In summary, then, we recognize that there are several behavioral factors such as diet, exercise, smoking, and marriage that are predictors of longevity; as are also several status characteristics such as sex, race, intelligence, and socioeconomic status. However, the explanations of why these variables predict longevity are usually ambiguous at best.

Various study designs that may help resolve these ambiguities and find other predictors of longevity include the use of the longevity quotient, longitudinal studies, and retrospective studies using records or previous surveys.

While such studies will not discover a "fountain of youth," they may help to unlock the secrets of longevity and make it possible to extend the proverbial "three score and ten years" to five score or more.

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Discussion

Kovar: Dr. Palmore, if you are using the national data for race, you may not be talking about blacks and whites; you may be talking about the white population as opposed to all others. It is a misleading generalization, if you are using data referring to nonwhites and calling them blacks. In the older ages, you are studying a crossover effect probably due, in part, to higher survivorship in the other nonwhite populations rather than to the black population.

Palmore: I was under the impression that there were some statistics on blacks only. (My review of the data found that they were comparisons of blacks—not all nonwhites—with whites.)

Siegel: You suggested that this crossover was hypothetical. A recalculation of the mortality rates of the older ages based on the Social Security Administration data sustains the crossover at a slightly higher age, more like at 80 to 84, rather than at 75 to 79. The merits of the Social Security data based on Medicare are that the numerator and the denominator come from the same basic collection system. Hence, there is not any secondary reporting of age as there is in computing vital statistics, where you get age from a death certificate for the numerator and age from the census for the denominator. I do not mean to imply that the Social Security data are completely valid either.

The crossover phenomenon occurs in other contexts that we do not understand. For example, it occurs in the death rates between the United States and Puerto Rico.

Greenhouse: Dr. Palmore, some of the statements you made had me concerned about their epidemiologic implications. Let me concentrate on one. I think you said something to the effect that an individual arriving at age 60 may have had some genetic factor which contributed to survival to age 60, but its effect disappears beyond age 60.

Palmore: Let me clarify that. To put it another way, those individuals who have some defective genes that would affect their mortality rates at an earlier age are wiped out and do not survive to age 60. The idea is that once you have made it to 60, this proves that you have a basically sound genetic substrata and there are no important genetic variations that now influence the remaining longevity of those that have survived that long.

Greenhouse: Well, I am not sure that I understand. Is the population above age 60 that homogeneous, which would permit you to say—with regard to behavioral factors, or disease factors—that there is no genetic variation which contributes to differences above age 60?

Palmore: I do not know. I am only saying that, in our study, we found no relationship between the longevity of our subjects and their parents. One possible explanation would be that at that age there is no longer any genetic effect.

Kasl: I have always been puzzled by the association of longevity with work satisfaction, especially among blue collar workers. I have found in younger age groups that work satisfaction is correlated with many other variables but to find it that predicts longevity is a little surprising.

I was wondering, is it a fairly clear relationship in the sense that older people like physical exercise? If you cannot do housework or hobbies like gardening, you do not report satisfaction. In other words, if you are already disabled these things are not enjoyable, either hypothetically or actually. Is that what is being tapped?

Palmore: Statistically, we attempted to control for their general health. When we entered in the first factor, that of physical functioning ratings, then work satisfaction still is a very strong variable. As you know, statistical control does not always completely control what you would like it to control. While physical functioning ratings diminish the relationship of work satisfaction somewhat, showing that there is an intercorrelation, the work satisfaction still contributes to substantial improvement for the prediction equation.

Kasl: So you do interpret it as a kind of morale influence?

Palmore: Yes, morale and an indicator of remaining active and doing something meaningful.

Atchley: We have been doing a longitudinal study of retired people. One of the things we found in our study was that health trend, i.e., whether the individual saw his health as staying the same, doing better or declining, had a direct impact on morale. It also had a direct effect on both the level of absolute activity and the individual's perception of having an activity deficit. So, health has a very strong impact on morale from two different sources.

Cardiovascular Risk Factors in The Aged: The Framingham Study

William B. Kannel, M.D., M.P.H.

Boston University
School of Medicine
80 Concord Street
Boston, MA 02118

Tavia Gordon

National Heart, Lung, and Blood Institute
National Institutes of Health
Bethesda, MD 20205

As the Framingham study's findings are at last being transferred into clinical and preventive applications among young and middle-aged adults, in an effort to reduce premature mortality (i.e. prior to age 65), the Framingham study cohort itself has entered the geriatric age group. It is not unreasonable to determine whether the same or other risk factors operate to promote cardiovascular disease in the aged as in the younger candidate. This may not be justified as a quest for immortality but it is not outside the realm of possibility that the correction of contributing factors, even in the aged, will result in a better quality of life.

Risk factors which promote cardiovascular disease are a constellation of influences. Some, such as those determining susceptibility (e.g. age, sex, and heredity) we can do little about. Atherogenic personal attributes such as serum lipids, blood pressure, and glucose tolerance are modifiable. Living habits that promote these traits such as faulty diet, overeating and physical inactivity are also modifiable. Other habits like cigarette smoking which can precipitate coronary attacks are also theoretically correctable. Signs of preclinical cardiovascular disease, including ECG abnormalities, cardiac enlargement and pulse wave abnormalities indicate impending cardiovascular disaster. These factors have been shown to be powerful contributors to risk under age 65, the risk mounting in proportion to the number and level of these contributing factors (figure II-4). The purpose of this report is to examine the utility of these and other risk factors for predicting cardiovascular disease in the elderly, and the implications for prevention of cardiovascular disease in the aged.

There is a common, if mistaken, notion that the usual cardiovascular risk factors no longer apply in older persons. There is some evidence that this may be so. The impact of serum cholesterol (table II-1) and cigarettes (table II-1) definitely diminishes with advancing age. For cigarettes there is no discernible effect after age 65 (table II-1) and, while there is a halving of risk in those who give up smoking before age 65, compared to those who continue to smoke, there is no discernible benefit after age 65 (figure II–5). Diabetes also appears to have a waning impact with advancing age, particularly in women (table II–1). Furthermore, the combined effect of these risk factors, even including hypertension, diminishes with advancing age (table II–2). There is only one major exception to the evidence of a diminishing impact of conventional risk factors with advancing age—blood pressure (table II–1). Compared to the other major identified contributors to cardiovascular disease in the aged, hypertension or blood pressure stands out as the major risk factor. This warrants a detailed examination of the role of blood pressure as a contributor to cardiovascular disease in the aged.

Table II-1.

Regression of incidence of cardiovascular disease on specified risk factors according to age and sex

	Univariate regression coefficients (unstandardized)					
	Diabetes		Hypertension		Serum cholesterol	
Age	Men	Women	Men	Women	Men	Women
45 to 54	0. 988	1. 806	0. 521	0. 654	0. 008	0. 008
55 to 64	. 701	1. 152	. 544	. 668	. 006	. 005
65 to 74	. 769	. 629	. 575	. 654	–. 000 [1]	. 004

	Cigarettes		Relative weight		ECG–LVH	
Age	Men	Women	Men	Women	Men	Women
45 to 54	0. 254	0. 009 [1]	0. 012	0. 016	0. 887	0. 960
55 to 64	. 188	–. 005 [1]	. 010	. 011	. 748	1. 015
65 to 74	–. 052 [1]	. 145 [1]	. 006	. 000 [1]	. 671	. 791

[1] Not significant.

Men and women 45 to 74. Framingham study: 20-year followup.

Hypertension

An examination of the prevalence of hypertension in the general population at time of initial examination at Framingham, before effective antihypertensive agents became available, indicates for the age range 45 to 64, about 25 percent afflicted (table II–3). Even at exam 10, after effective antihypertensive therapy became generally available, hypertension is still highly prevalent and rises with advancing age (table II–3). Even today, about every third person over 65 has hypertension.

Table II-2.

Age trend in incidence of cardiovascular disease—8-year probability of cardiovascular disease (%)

Age	Men		Women	
	Optimal risk	Poor risk	Optimal risk	Poor risk
35	0. 6	60. 2	0. 4	19. 5
40	1. 2	70. 8	0. 7	28. 4
45	2. 2	77. 8	1. 3	38. 0
50	3. 7	81. 9	2. 2	47. 0
55	5. 5	84. 1	3. 4	54. 7
60	7. 4	84. 8	5. 1	60. 6
65	9. 0	84. 0	7. 0	64. 9
70	10. 0	81. 7	9. 0	67. 5

	Risk category	
	Optimal	Poor
Systolic B.P	105	195
Cholesterol	185	335
Glucose intol	0	+
Cigarettes	0	+
ECG–LVH	0	+

Men and women 35 to 70. Framingham study: 18-year followup.

An evaluation of age trends in blood pressure reveals a disproportionate rise in systolic pressure with advancing age compared to that in diastolic (figure II–6.1). In cross-sectional data women's pressures appear to be distinctly lower than men's initially, rising to meet those of men at middle age and crossing over thereafter to end up higher. In cohort data, where pressures are followed as people actually age, pressures never do cross over in the sexes, with women's systolic pressures starting lower and gradually converging on those of men. Diastolic pressures remain essentially parallel at a lower level in women than men, reaching a peak at around age 60 and declining thereafter (figure II–6.2). The discrepancies between cross-sectional and prospective data possibly derive from differential mortality in men and women.

Because blood pressures increase with age, some appear to contend that this is either a physiological sign of progressive increase in arterial rigidity or a compensatory change to ensure tissue perfusion through narrowed vessels. However, there is no indication that hypertension is more benign in the aged than the young (table II-4). Neither the absolute nor relative risk is any lower in the elderly than the young hypertensive. Even borderline elevations of pressure in the elderly double the risk (table II-4). Even for systolic blood pressure, gradients of risk of cardiovascular sequelae show no indication of a waning impact with advancing age (figure II-7). There is also no indication that risk of cardiovascular disease in the elderly is more closely related to the diastolic than the systolic component of the blood pressure (table II-5).

Table II-3.
Prevalence of hypertension at exams 1 and 10 by age and sex

Age	Percent HBP[1]			
	Exam 10		Exam 1	
	Men	Women	Men	Women
45 to 49	15	6	20	19
50 to 54	17	9	27	29
55 to 59	16	16	25	36
60 to 64	20	24	32	39
65 to 69	22	25	1	1
70 to 74	20	37	1	1
75 to 79	24	43	1	1

[1] HBP = ≥160/95
Men and women 45 to 79. Framingham study.

Table II-4.
Risk of cardiovascular disease according to hypertensive status and age

Hypertensive status	Average annual incidence per 1,000					
	Men			Women		
	45-54	55-64	65-74	45-54	55-64	65-74
Normal	8. 6	15. 6	17. 1	2. 7	6. 1	8. 6
Borderline	14. 5	30. 4	32. 7	6. 1	14. 4	22. 5
Hypertension	23. 6	43. 9	51. 0	9. 7	23. 7	35. 6

Men and women 45 to 74. Framingham study: 20-year followup.

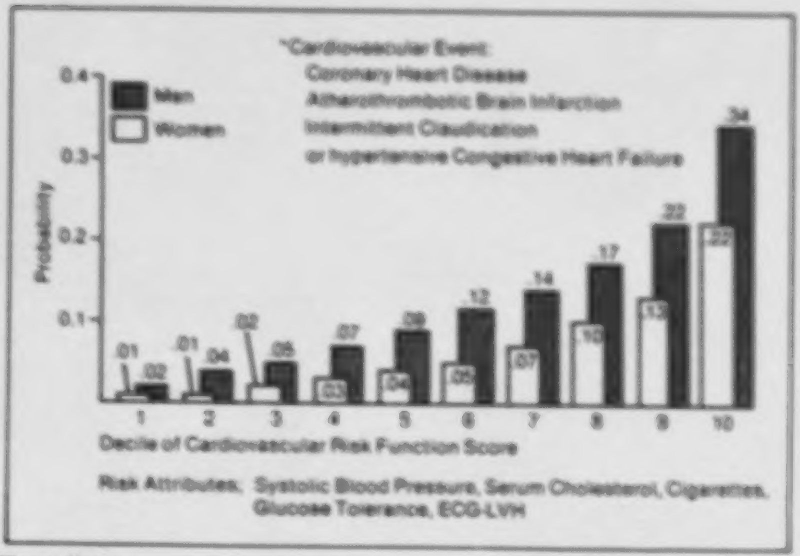
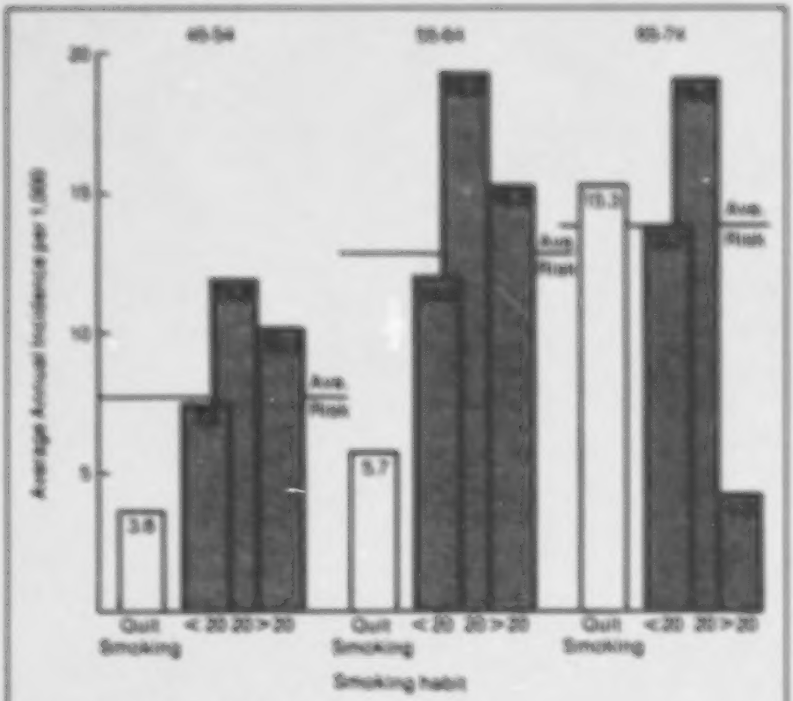


Figure II–4.
Probability of a major cardiovascular event* in 8 years according to risk function score. Men and women 35 to 74. Framingham study: 18-year followup.

Thus, there is no indication that the disproportionate rise in systolic pressure with advancing age is innocuous and even at low diastolic pressures, risk rises in proportion to the accompanying systolic pressure (figure II–8). There is also no evidence that women in advanced age tolerate hypertension better than men. The attributable risk for women for overall mortality, for cardiovascular mortality and for cardiovascular morbidity is actually greater for women over 65 than men (table II–6). In fact, for women there is no indication of a decreasing attributable risk for hypertension with advancing age as there appears to be for men for overall and cardiovascular mortality (but not morbidity) (table II–6).

It is not safe in elderly hypertensives to await the appearance of evidence of target organ involvement before treatment since half the cardiovascular sequelae appear before such evidence can be detected on biennial examinations (table II–7).

Labile blood pressure elevations are considered less serious than fixed elevations. This is a logical fallacy since all blood pressures are labile and high pressures are more labile than low ones (figure II–9). Lability of pressure is also more pronounced in the elderly than the young (figure II–10). It is not safe to judge the need for treatment based on the lowest pressure recorded on a patient if the average pressure is high, even in the elderly.



	45-54	55-64	65-74
Regression Coeff.	0.340	0.317	-0.173
Standard Error	0.109	0.128	0.228
T Value	3.12	2.47	-0.77

Figure II-4.
Incidence of coronary attacks among cigarette smokers according to subsequent cigarette habit. Men 45 to 74 at exam. Framingham study: 18-year followup.

Lipids

An examination of the impact of the serum total cholesterol at different ages shows a definite waning effect with little impact after age 65 (table II-1). Cholesterol is partitioned in three major lipoprotein fractions—LDL, HDL and VLDL. Examination of the risk of cardiovascular disease according to the cholesterol-lipoprotein fractions reveals an impact of cholesterol in the HDL and to a lesser extent in the LDL fraction (table II-8). That in the VLDL fraction seems to have little effect. Triglyceride appears to have little impact in men beyond age 50, while there is an effect in women up to age 70, but this too disappears when the other lipids and risk factors are taken into account (table II-8).

Table II-3.
Risk gradients according to systolic vs. diastolic blood pressure

Blood pressure component	Standardized regression coefficients							
	CHD		CHF		OPAD		CVA	
	Men	Women	Men	Women	Men	Women	Men	Women
Systolic	0.207	0.417	0.302	0.303	0.051[1]	0.432	0.317	0.620
Diastolic	.214	.200	.270[1]	−.027[1]	−.270[1]	.167[1]	.271[1]	.322

	Total C-V	
	Men	Women
Systolic	0.294	0.643
Diastolic	.130[1]	.226

[1] Not significant at p < .05.
Men and women 45-74. Framingham study: 18-year followup.

Table II-4.
Attributable risk for hypertension according to age

	Over-all mortality	Cardiovascular mortality	Cardiovascular morbidity
Men			
45 to 54	17.9	28.3	16.4
55 to 64	16.1	21.4	17.9
65 to 74	8.4	12.9	18.6
Women			
45 to 54	12.0	28.6	17.4
55 to 64	8.5	17.5	28.5
65 to 74	14.9	34.5	26.9

Attributable risk = Total pop. rate − Non-HBP rate/Total pop. rate X 100.
Men and women 45 to 74. Framingham study: 18-year followup.

Table II-7.

Incidence of cardiovascular disease in hypertensives prior to development of target organ involvement [1]

	Number hypertensive and free of C-V disease	Number developing C-V disease without prior abn.	Percent of C-V cases free of prior abn.
MEN			
35 to 44	90	18	78.3
45 to 54	104	19	45.2
55 to 64	42	19	45.2
Total	235	56	52.3
WOMEN			
35 to 44	30	4	30.8
45 to 54	152	23	41.7
55 to 64	175	29	39.2
Total	357	52	40.9

[1] ECG Abn.—LVH, IVB, NSA, and OCE on X-ray. Exam. 3-10.
Men and women 35 to 64. Framingham study: 16-year followup.

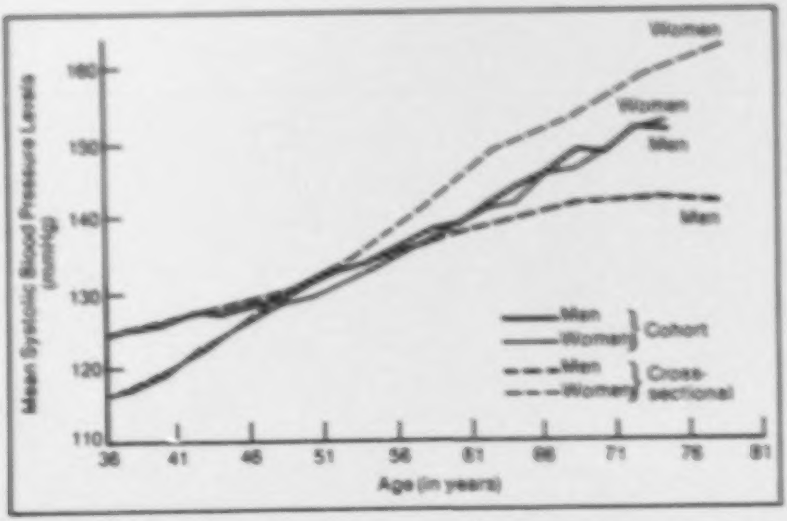


Figure II-6.1.

Average age trends in systolic blood pressure levels for cross-sectional and cohort data. Men and women. Framingham study: Exams 3-10.

From: Gordon, T. and Shurtleff, D.: Means at each examination and inter-examination variation of specific characteristics. Framingham study, exam 1 to exam 10. *The Framingham Study, Section 29.* Wash., D.C., U.S.G.P.O., 1974.

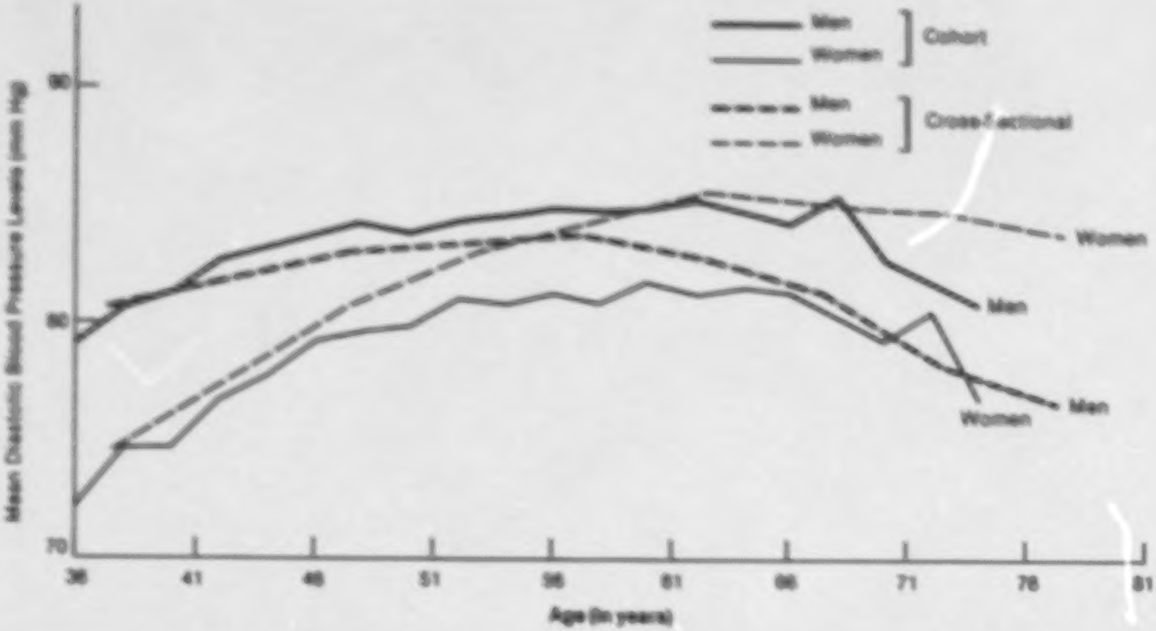
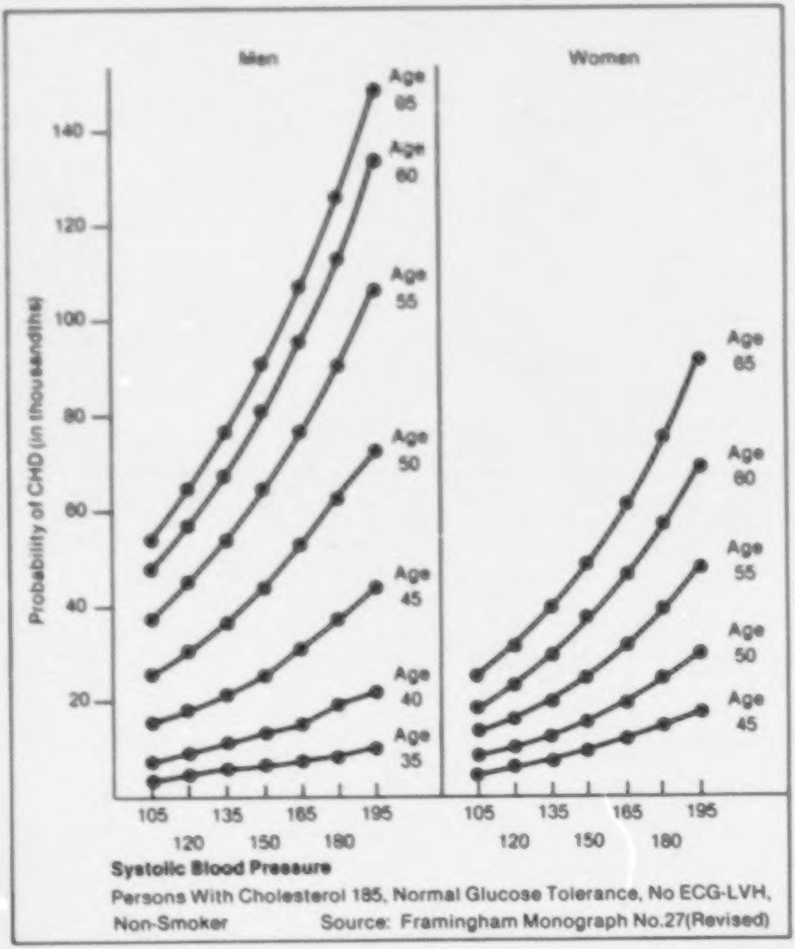


Figure II-4.2.
Average age trends in diastolic blood pressure levels for cross-sectional and cohort data. Framingham study: exams 3–10. From: Gordon et al., 1974.

Of all the lipids and lipoproteins, HDL-cholesterol has the greatest impact on risk in old age (table II-8). A strong *negative* association between HDL-cholesterol and CHD incidence is noted in both sexes on up to age 80 (table II-9). This effect can be demonstrated even after adjustment for the coexisting effect of the other lipids and other risk factors (table II-9).

Glueck and associates in a study of long-lived families have also noted that they tend to have unusually high HDL values (*1*). It is



Persons With Cholesterol 185, Normal Glucose Tolerance, No ECG-LVH, Non-Smoker   Source: Framingham Monograph No.27(Revised)

Figure II-7.
Probability of developing coronary heart disease in 6 years according to systolic blood pressure. Low risk persons 35 to 65. Framingham study: 16-year followup.

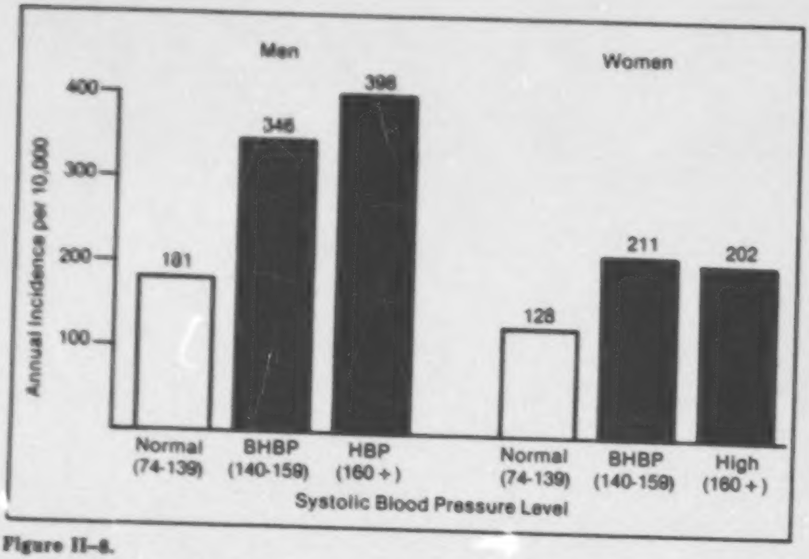


Figure II-8.

Risk of cardiovascular disease according to systolic blood pressure at diastolic blood pressure less than 90 mm Hg. Men and women 65 to 74. Framingham study: 18-year followup.

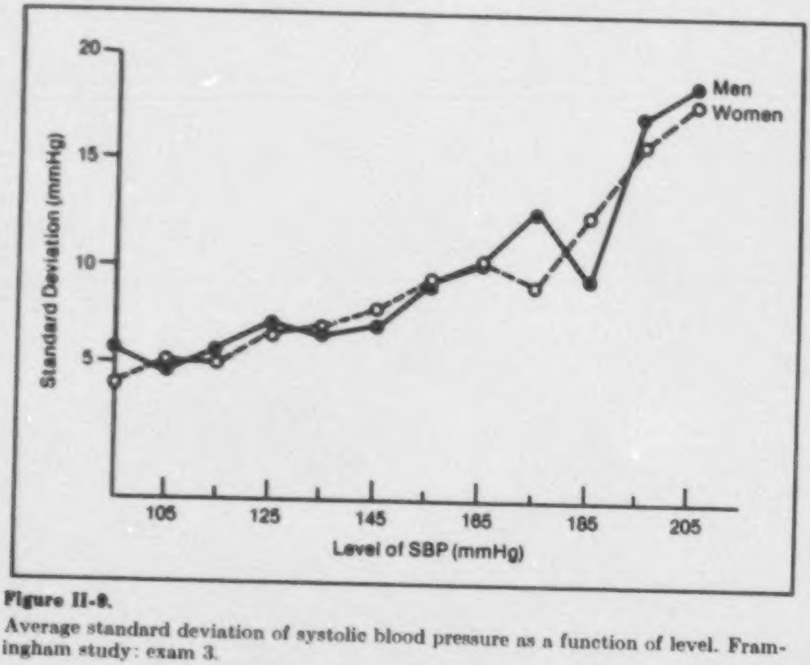


Figure II-9.

Average standard deviation of systolic blood pressure as a function of level. Framingham study: exam 3.

Table II-8.
Standardized logistic regression coefficients for CHD incidence

Characteristics[1]	Standardized logistic regression coefficients[2]			
	Univariate		Multivariate	
	Men	Women	Men	Women
HDL cholesterol	−0.488[5]	−0.741[5]	−0.610[5]	−0.650[5]
LDL cholesterol	.288[3]	.303[3]	.332[4]	.260[3]
Triglyceride	.048	.276[4]	−.092	−.106
Stystolic blood pressure	.323[4]	.400[5]	.327[5]	.216
ECG–LVH	.279[5]	.207[4]	.245[4]	.150[3]
Relative weight	.029	.283[3]	−.016	.031[3]
Diabetes	−.024	.474[5]	−.114	.390

[1] Measured at exam 11.
[2] Estimated by method of Walker-Duncan.
[3] $p < .05$ [4] $p < .01$ [5] $p < .001$
Men and women 49 to 82. Framingham study: exam 11.

Table II-9.
Univariate and multivariate logistic regression coefficients for CHD on HDL cholesterol

Age and sex	Number of cases	Univariate	Multivariate	
			Lipids	Lipids and other factors
Men				
50 to 59	25	−0.496[1]	−0.543[1]	0.618[1]
60 to 69	32	−606[2]	−.664[2]	−.720[1]
70 to 79	22	−.495	−.495	−.499
Women				
50 to 59	27	−.680[2]	−.640[2]	−.922[2]
60 to 69	19	−1.318[3]	−1.019[3]	−.770[1]
70 to 79	17	−.087	−.132	−.261

Coefficients are estimated by the method of Walker-Duncan and standardized. Lipids include LDL cholesterol and triglyceride. Other factors refer to systolic blood pressure, LVH–ECG and relative weight.
[1] $p<.05$ $(t>1.65)$ [2] $p<.01$ $(t>2.33)$ [3] $p<.001$ $(t>3.09)$.
Men and women 50 to 79. Framingham study: exam 11.

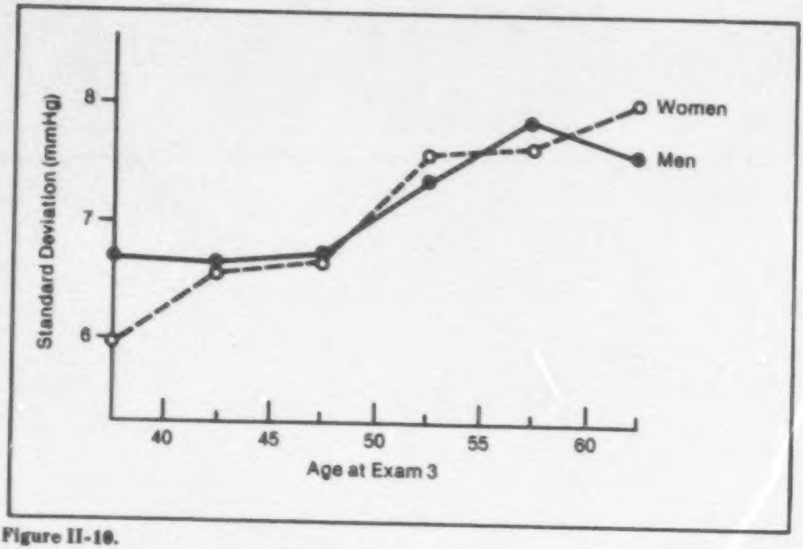


Figure II-10.
Intra-individual standard deviation of systolic blood pressure by age and sex. Framingham study: exam 3.

conjectured that HDL serves as a scavenger lipoprotein facilitating removal of cholesterol from deposits and interfering with cellular uptake of excess cholesterol (*2–4*).

Cardiovascular Risk Profiles

Using ordinary office procedures (blood pressure, ECG, and a cigarette history) and simple laboratory tests (a casual blood test for sugar and cholesterol) it is possible to estimate risk of a variety of major cardiovascular events over a wide range (figure II–4). By synthesizing this information into a composite risk estimate using a multiple logistic function (*5*), risk can be efficiently estimated detecting those with multiple marginal abnormalities at high risk as well as those with marked elevations of single risk factors.

Because several components of the risk profile (cigarettes, cholesterol and glucose tolerance) have a minimal impact over age 60 it seems reasonable to seek more potent ingredients from which to construct a risk profile for the aged. Of 1,025 men and 1,445 women aged 49 to 82 and free of CHD at the 11th biennial examination, 79 men and 63 women subsequently developed CHD in the Framingham cohort. Using a risk function based on HDL and LDL cholesterol, systolic blood pressure, ECG–LVH and diabetes, less than 2 percent of the subsequent CHD cases were found in the lowest decile whereas 25 percent of the cases for men and 37 percent for women were found in the highest decile (figure II–11). Predictability held for each specific age group over 50. This predictability was at least as good as that obtained by the usual CHD risk profile at younger ages using the conventional risk factors. It thus appears possible to select candidates for cardiovascular disease in advanced age as well as in the young. Since some cardiovascular risk factors are applicable at older ages, the possibility of deferring cardiovascular disease at this stage in life becomes more plausible.

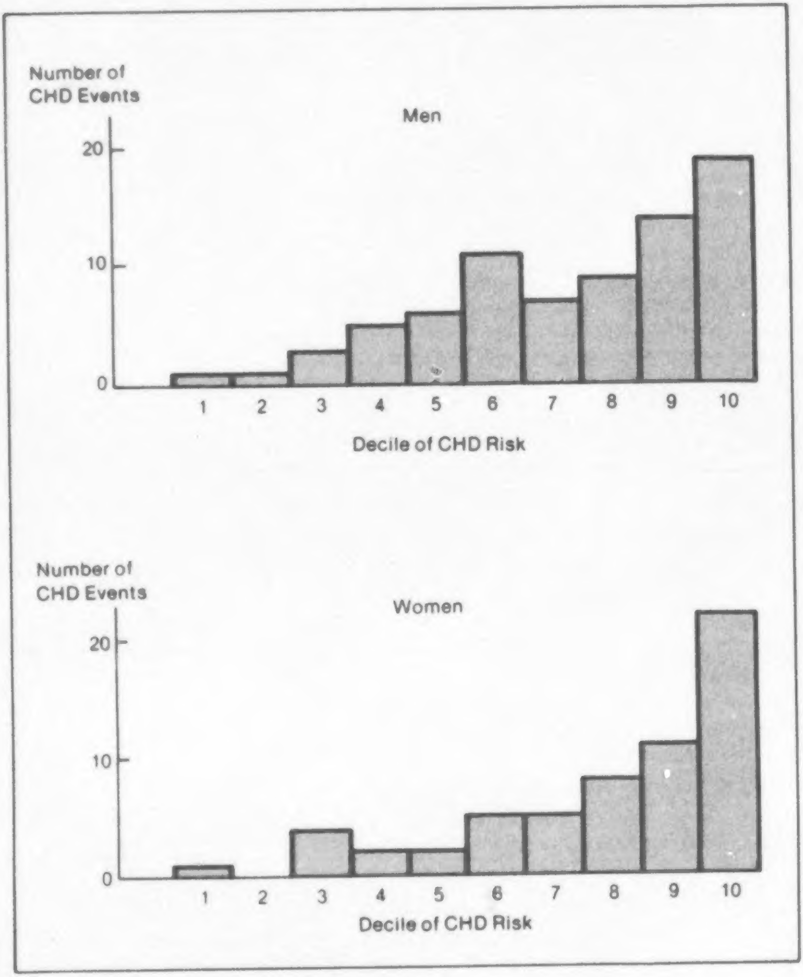


Figure II–11.
Incidence of CHD by decile of risk. Framingham study: exam 11.

In the older age group HDL-cholesterol, LDL-cholesterol, triglyceride, systolic blood pressure, relative weight, diabetes and ECG-LVH are, taken alone, associated with CHD risk (table II–8). When the net effect of each is assessed by combining them into a multivariate risk function, triglyceride and overweight no longer contribute significantly to the observed rate of occurrence of cardiovascular disease. The relation of triglyceride to risk of CHD, more apparent in this age group in women than men, seems to be a consequence of its association with diabetes and low HDL-cholesterol levels. Overweight is also a noncontributor in conjunction with the other risk factors but its frequent association with elevated blood pressure, depressed HDL-cholesterol and diabetes makes weight control an important feature of any prophylactic program. It is not clear whether control of triglyceride per se improves the impaired glucose tolerance, low HDL-cholesterol and overweight, with which it is often associated.

Although it is repeatedly observed that the total serum cholesterol is, at older ages, not related to CHD risk, it is now clear that two of its components—HDL and LDL—are acting in opposite directions. By analyzing the serum cholesterol for these separate components, it is possible to demonstrate that each is associated with cardiovascular risk in the elderly. Whether correcting this pair at this stage in life is beneficial remains to be demonstrated.

In any event, it is possible in this older cohort of the Framingham study to segregate low risk from high risk coronary candidates, over at least a fifteen fold range, comparing lower and upper quintiles. CHD incidence at advanced ages is no more a random process than it is at younger ages. Since most of the risk attributes which are predictive are modifiable there is some possibility that CHD risk is also modifiable, even at this age. Of course, ability to predict is no guarantee that interventions to correct the ingredients of the risk profile will necessarily be fruitful. However, it would seem worthwhile to try and find out in order to obtain a gain in the quality, if not the quantity of life in later years.

Aging and Cardiovascular Disease

It is difficult to specify precisely what aging is. It is likely more than the sum of failing eyesight and hearing, slowed reflexes, loss of stature, gnarled stiff joints, wrinkled skin, loss of vigor, strength, and memory, poor smell and taste, or even impaired cardiac function.

However, cardiovascular disease is a prominent feature of growing old and is too often the undeserved reward for achieving a venerable stage in life. Strokes, cardiac failure and occlusive peripheral arterial disease are all too often a feature of growing old and are seldom seen prior to age 60 (table II–10). Also, as in the young, coronary disease is a common affliction in the elderly. There is also some reason to believe that progressive shutting down of the circulation due to atherothrombotic, embolic and arteriolar disease may contribute to the general decline in organ function with advancing age. In any event, less cardiovascular disease would certainly make old age easier to bear. Avoidance of strokes, in particular, which rob its victims of their ability to communicate, their dignity and independence, and physical prowess and self-reliance would be most welcome.

Table II–10.
Incidence of cardiovascular events according to age and sex

Age	Average annual incidence per 1, 000							
	Coronary heart disease		Cerebrovascular accident		Peripheral arterial disease		Congestive heart failure	
	Men	Women	Men	Women	Men	Women	Men	Women
45 to 54	9. 9	3. 1	2. 0	0. 9	1. 8	0. 6	1. 8	0. 8
55 to 64	20. 8	9. 5	3. 2	2. 9	5. 1	1. 9	4. 3	2. 7
65 to 74	20. 4	14. 5	8. 4	8. 6	6. 3	3. 8	8. 2	6. 8

Men and women 45 to 74. Framingham study: 20-year followup.

An examination of the gradient of risk with advancing age in the Framingham cohort reveals a steep rise even in those who are entirely free of cardiovascular risk factors (table II–2). It is also of interest, however, that the age trend in cardiovascular disease incidence is much less steep in those who are in the worst cardiovascular risk category, indicating that cardiovascular risk factors can blunt the effect of age. It is also clear that even over age 65 those with multiple risk factors are at greatly increased risk compared to those free of them, although the *relative* risk is less pronounced than in the young. It appears that multiple risk factors can make one old beyond one's years as regards the cardiovascular apparatus.

The change in biomedical features of the cardiovascular and cerebrovascular systems with age is not only difficult to ascertain, but hard to conceptualize as well. The chief difficulty is to dissociate those changes due to aging per se from those due to some environmental or internal noxious influence acting overtime. The mere fact that the incidence of a condition increases with age does not establish it as a direct consequence of aging. In evaluating the pathology attributable to aging it is necessary to establish some unique effect. At least three possibilities must be considered: 1) senescence of tissues per se, 2) altered ability to cope with ever present noxious influences, 3) the greater time of exposure to these noxious agents in the aged.

Using multivariate analysis which attempts to take age into account for each of the risk factors indicates that age does indeed account for a part of the effect of each factor on cardiovascular disease incidence (table II–11). It is true for every cardiovascular disease end point and even when all factors are considered together. This is one way of trying to sort out whether age is exerting a unique effect. However, even here we cannot be sure that the residual risk assigned to age is reflecting more than the time-dose product of the risk factors—age meaning how many years exposed to the risk factor.

Table II–11.
Regression of incidence of cardiovascular disease on specified risk factors with and without age

Regression coefficient	Diabetes		Hypertension		Serum cholesterol	
	Men	Women	Men	Women	Men	Women
Univariate[1]	0.907	1.313	0.574	0.803	0.005	0.008
Bivariate[2]	.766	1.025	.538	.643	.006	.006

Regression coefficient	Cigarettes		Relative weight		ECG–LVH	
	Men	Women	Men	Women	Men	Women
Univariate[1]	0.107	−0.111	0.009	0.011	0.839	1.098
Bivariate[2]	.189	.053	.010	.010	.750	.916

[1] Factor alone.

[2] Factor plus age.

Men and women 45 to 74. Framingham study: 20-year followup.

Some of the phenomena we associate with aging like baldness, graying hair, presbyopia and loss of stature are not related to cardiovascular disease incidence at a given age. Is it aging per se that causes cardiovascular incidence and risk factors to increase with age, or only the time-dose product of the action of identifiable risk factors?

Preventive Measures

So far as preventive measures are concerned, it must be recognized that cardiovascular disease is an insidious disorder which has its roots in young adult life, possibly even in childhood. Logically, then, preventive measures should be applied early in life to reduce the burden of these diseases both in the elderly and young candidates. However, this does not necessarily mean that all preventive measures are worthless once a venerable stage in life has been reached.

Conclusions based on interventions in the young or middle-aged are not necessarily applicable to older people. The absolute risk in the elderly is considerably higher than in the young. The subclinical pathology in the aged is likely to be more severe and hence more difficult to regress. Once old age is attained some risk factors have relatively little impact. Those escaping disease to advanced age may be less susceptible to the known risk factors and better equipped biologically to cope with them.

These considerations suggest that preventive efforts beginning in the elderly may have little value. However, we must not lose sight of the fact that because of the relatively high incidence of mortality in the elderly the *absolute* impact of preventive measures short-term may actually be greater in the elderly than the young despite a lesser *relative* impact. Also, since life expectancy is short in the elderly, freedom from cardiovascular disasters could enhance the quality of the remaining years of life, if not its duration. A small reduction in morbidity and mortality over 5 years in an elderly population may save more lives and produce a greater reduction in suffering than a much greater reduction in the same number of younger candidates for cardiovascular disease.

There is some indication that cardiovascular mortality is not a fixed liability of aging. Examination of secular trends in cardiovascular mortality over the past decade reveals a declining coronary and cerebrovascular mortality. This improvement is not confined to the young and is also apparent in those over age 65 (table II–12). This would appear to indicate that there are modifiable contributors worthy of attention. Although this does not eliminate the need for controlled preventive trials in the aged, it does justify some optimism.

Table II–12.
Percent change in heart attack death rates, U.S. 1968–74

Age group	Rate per 100,000			
	White		Nonwhite	
	Men	Women	Men	Women
35 to 44	−21	−20	−31	−37
45 to 54	−13	−14	−16	−22
55 to 64	−15	−15	−18	−26
65 to 74	−14	−20	−26	−30

8th revision, codes 410–413.
Source: *American Heart News*, vol. III, No. 8, 1976.

The specific preventive measures which may be of value in the elderly are first and foremost blood pressure control. Also, of potential benefit are weight reduction, control of HDL-cholesterol and possibly, encouragement of physical activity.

In the present state of our knowledge (or ignorance), it seems clear that the key correctable risk factor for cardiovascular disease in the aged is hypertension. This is one factor which is a powerful contributor to all the major cardiovascular diseases which afflict the elderly: it is one factor which operates with as much force in the aged as in the young; it is correctable and easily detected; it is highly prevalent in the aged; and there is already evidence that correction of hypertension is effective in reducing overall mortality and the incidence of strokes and congestive heart failure (*6*, *7*). This needs greater emphasis because an examination of treatment of hypertension by age and blood pressure level indicates that only a small percentage of hypertensives receive treatment and the percentage receiving treatment decreases with advancing age (table II–13). Even at 200 mmHg systolic blood pressure, less than 32 percent receive treatment.

Table II–13.
Percent receiving antihypertensive drugs

Systolic Blood Pressure [1]	Age			
	35–44	45–54	55–64	65–74
MEN				
140 to 149	2. 26	2. 23	2. 49	1. 66
150 to 159	7. 75	5. 74	7. 12	7. 02
160 to 169	10. 42	8. 25	8. 55	7. 02
170 to 179	12. 50	18. 89	15. 44	14. 81
180 to 189	23. 08	27. 27	23. 75	14. 29
190 to 199	25. 00	26. 92	30. 61	17. 39
200 plus	40. 00	27. 78	36. 73	20. 83
WOMEN				
140 to 149	3. 45	5. 30	5. 86	5. 95
150 to 159	10. 53	10. 84	6. 35	7. 97
160 to 169	10. 71	14. 76	11. 60	9. 47
170 to 179	22. 22	20. 77	16. 28	16. 13
180 to 189	22. 22	31. 43	17. 45	20. 25
190 to 199	40. 00	33. 33	40. 85	26. 92
200 plus	42. 85	53. 33	39. 58	32. 26

[1] On exam preceding the first use of antihypertensive drugs.
Men and women 35–74. Framingham study: exams 4–10.

This low treatment rate derives from not only difficulty in attaining adherence to treatment, but from a number of misconceptions some physicians appear to have about hypertension. Elevated blood pressure is not a physiologic concomitant of aging; it is not less serious in the aged; systolic hypertension is not innocuous; the lowest pressure recorded is not the safest pressure on which to judge the need for treatment; and it is not safe to await target organ involvement before treating.

Successful control of multiple risk factors requires personalized care including counseling and long-term treatment to control risk factors, in addition to surveillance to detect and monitor changes in risk factors.

Other systems of health care than the ordinary primary practitioner may be required to accomplish this because of the size and nature of the problems, the need for frequent followup, and the competition of medical illness which must be given priority.

Intervention programs to modify risk attributes in the elderly constitute a formidable problem because of the high prevalence of the contributors to cardiovascular disease in the elderly (table II–14). Most are exposed to at least one risk factor, and even confining attention to hypertension, we must contend with 27–48 percent of the aged population. Risk profiles can assist in selecting those in greatest jeopardy for preventive medical interventions. However, because of the high prevalence of candidates for cardiovascular disease, personal hygiene and public health measures are also required to reduce overweight, increase physical activity and modify diet. Preventive medical interventions should place particular emphasis on management of hypertension. Treatment should be kept as simple as possible, to en-

Table II–14.

Percent prevalence of selected "risk factors" in the United States

Age and sex	Inactivity	Obesity	Hypertension	Cigarette smoking	Diabetes	Hypercholesterolemia	ECG-LVH [1]
Men							
35 to 44	12. 1	12. 5	13. 5	48. 6	1. 1	20. 2	2. 9
45 to 54	16. 9	14. 7	18. 3	43. 1	1. 1	15. 7	4. 8
55 to 64	21. 0	12. 5	22. 3	37. 4	3. 3	23. 5	10. 1
65 to 74	27. 1	12. 7	27. 1	22. 8 [2]	3. 2	21. 6	7. 1
Women							
35 to 44	13. 3	20. 1	8. 5	38. 8	. 8	12. 9	. 9
45 to 54	19. 3	24. 2	18. 2	36. 1	2. 9	28. 0	3. 6
55 to 64	30. 8	30. 9	31. 2	24. 2	3. 2	49. 7	4. 1
65 to 74	39. 0	27. 2	47. 6	10. 2 [2]	6. 1	51. 9	9. 6

[1] Framingham, Mass.

[2] 65 and over.

Definitions: Inactivity is average oxygen consumption less than 0.30 liter/minute (1954–58); Obesity is weight 20 percent or more above median (1960–62); Hypertension is a blood pressure at least 160/90 (1960–62); Cigarette smoking refers to current habits (1970); Diabetes is medically treated (1960–62); Hypercholesterolemia is a serum cholesterol at least 260 mg% (1960–62); ECG–LVH is an electrocardiographic pattern (1948–53).

hance compliance; a modest reduction in pressure is better than none and normalization of the pressure should not be regarded as an indication for discontinuing treatment.

Effective control of cardiovascular risk factors in the aged requires a change in the emphasis and attitudes of physicians and other health workers. Old age is not a sin and the aged deserve preventive management as well as the young. The occurrence of a stroke, congestive failure or a coronary attack is not a just reward for achieving old age; nor is it an inevitable consequence of aging.

Recommendations

In conclusion, the following recommendations are made:

1) Further research on the operation of cardiovascular risk factors to determine which ones influence cardiovascular morbidity in the aged.
2) Clinical trials to determine the efficacy of hypertension control, in general, and control of systolic hypertension in particular, in the aged, since hypertension is the key remediable factor promoting cardiovascular disease in the elderly.
3) Studies to determine the amount and nature of the disability in the aged attributable to cardiovascular disease.
4) Studies of the reasons for poor compliance with antihypertensive therapy in the elderly are urgently needed in view of the high prevalence of untreated hypertension in this age group.
5) Studies of the efficacy of early detection and treatment of congestive heart failure versus treatment after overt signs appear warranted in view of the severe morbidity and poor prognosis found in the Framingham cohort.
6) Further clarification of the role of diabetes in promoting cardiovascular disease in the elderly is needed to determine if obesity-related keto-resistant, hyperinsulinemic diabetes exerts any unique effect.
7) The unique relation of HDL-cholesterol to cardiovascular disease at advanced age deserves further attention to learn more about the mechanism, what determines its level in the aged and how it can best be modified.

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Discussion

Weksler: I was interested in your statements that these lower blood pressure elevations, let us say 90—or even less than 90—to 105 were effectively treated. I thought Fries' study suggested that we could intervene only at the higher end and alter the natural history of the disease. Are these new data?

Kannel: The data that we are talking about here relate to the hazards of a given level of pressure and to the advocacy of treating it effectively.

Weksler: So you do not mean for us to conclude from your data that we can reduce the incidence of complications by treating patients who are borderline.

Kannel: I do not think that you can infer that necessarily from these data. However, as I pointed out, at 200 millimeters of mercury, systolic blood pressure, only about 30 percent of all elderly people receive treatment. Now those are the kind of pressures that Fries is talking about.

Palmore: Please explain the data about the decline in mortality.

Kannel: Lately, there has been reported a decline in overall mortality and in cardiovascular mortality, particularly between 1968 and 1975. The question has arisen whether this trend is in fact real and whether it is attributable to changes in life-style. Parenthetically, it is encouraging to see that even in the oldest age groups there was a significant decline in mortality.

Costa: Dr. Kannel, I found it very instructive that your data seemed to show a differential salience of risk factors. Rather than assuming that there is a continuing contribution from the other risk factors with age, only systolic blood pressure held up in the longitudinal analyses.

Kannel: Of the conventional risk factors, the one factor that seems to hold up best with advancing age seems to be blood pressure. We are looking at other factors as well. It looks like the lipids do contribute contrary to what we previously thought. We had been confining our attention to the total serum cholesterol. More detailed analyses of serum cholesterol reveals that there are two components involved, one the low density lipoprotein (LDL) cholesterol, which seems to be atherogenic and promotes this disease, the other high density lipoprotein (HDL) cholesterol which seems to be the transporter of cholesterol out of tissue and is therefore protective. There appears to be a continuing association of each lipoprotein fraction, particularly HDL cholesterol, with cardiovascular mortality and morbidity rates into the advanced years.

Costa: Do you have any speculations as to the declining influence of diabetes in the elderly?

Kannel: This problem deserves much more attention. It is an obesity-related, keto-resistant, hyperinsulinanemic kind of "diabetes." As such this may not have the same significance as an insulin dependent early age of onset type of diabetes. Investigators in the diabetes field lose sight of the fact that what they are talking about basically in their studies is hyperglycemia and that the assumption that this is a uniform entity, I think, is a gross oversimplification. The assumption that the solution to the problem is solely to reduce the blood sugar is also a misconception. Diabetes is a complex metabolic disorder, characterized not only by impaired glucose tolerance, but by lipid aberrations, overweight, and high blood pressure, all of which need attention. Perhaps, if the concept of control included the other metabolic aberrations, then we might see some change in the cardiovascular sequelae of diabetes.

Greenhouse: Do you think it is possible to obtain information relating longevity in parents with longevity of the index case? Do records for subjects in the Framingham study go back far enough to correlate data on risk factors to index cases with those risk factor measurements which might be obtained from records of parents?

Kannel: We do have subjects in this study who have siblings in the cohort and, lately, we have completed an offspring study. It is therefore possible to look at the level of risk attributes in the offspring of this cohort at the same age and stage in life as the parents. These are of the same genetic pool. This was done by Dr. Feinleib who found that certain risk attributes are lower at a given age in the offspring than they were in the parent.

We also studied the correlation of levels between parents, offspring, sibs, and spouses. Although we gained some insight, it is still very difficult to dissociate genetic from environmental influences. We are also in a position to look into the risk attributes of long-lived people in relation to those short-lived. In fact, many years ago, when these risk factors were systematically measured in the whole cohort, we included this capability in our planning.

Atchley: In the findings that you presented about the high density and low density cholesterol, how does that relate to the blanket treatment of trying to lower all kinds of cholesterol?

Kannel: Fortunately, those things that lower LDL cholesterol raise HDL. In other words losing weight lowers LDL some and raises HDL; exercise raises HDL. The kind of diet recommended by the Heart Association of low cholesterol, low saturated fat and reasonable calories, has the appropriate effect on HDL. Where this does become a problem, I think, is in the drug treatment. This may explain some of the drug treatment failures in that little attention has been paid to what has happened to HDL while trying to reduce LDL.

Wilkie: In defining hypertension, what would you call your lower limits for older and middle-aged people or is it the same at all ages?

Kannel: We have looked at this in two ways. We looked at this in terms of hypertension as viewed by clinicians, because they think categorically, but we think this is nonsense. We also looked at it in terms of blood pressure levels. The data you saw are the regression of the incidence of cardiovascular disease on blood pressure level and these regressions seem to hold up pretty well with advanced age. I do not know what a normotensive blood pressure level is in an old person compared to a young one. I feel it is not a meaningful question. In the short term, the absolute risk is higher in the elderly at any level of pressure and the relative risk is as great as in the young.

Wilkie: There has been some work indicating that cardiovascular disease and hypertension are related to the rate of intellectual impairment among middle-aged and older people. This is getting to be an issue now. At what point do you try to classify your people?

Kannel: I would recommend looking at blood pressure without any preconceived notions as to categorical hypertension. I recommend just looking at the intellectual scores and various parameters of intellectual function according to blood pressure level without worrying about what is being designated as hypertension.

Siegel: Your chart showed 1968 to 1975 as the period with the declining trend of cardiovascular disease.

Cohen: Is that the same for men and women?

Kannel: The total mortality for cardiovascular disease seems to be down in both sexes and in all age groups. It is not just a result of a regrouping phenomenon which you often run into with different fashions or changes in death certification. It is a real phenomenon and whether it is due to changes in life-style is, of course, speculative, but encouraging.

Waldron: If we look at death rates from 1900 on, we see a tendency for them to rise during each economic boom and to fall during each recession. This indicates that the recent decline in death rates may be related to the recession in the economy.

Kannel: We gain credibility only if we see a sustained effect in mortality--a real trend—and if we are sure that we have not been confused by change in fashion in death certification. I would say, anytime we see a fall in the cardiovascular mortality, we should see a fall in overall mortality. If these two are disassociated, I would not believe the data at all.

Kovar: I was interested in some of your statements about people at the older ages, particularly with regard to the sex differences. Since mortality for cardiovascular diseases is higher for males at younger ages, have you used life table analysis to see what happened in the kind of population you had?

Kannel: The gap in incidence closes with advancing age. We have looked to see if we could explain the favorable experience of women compared to men on the conventional risk factors and we cannot. Are you suggesting that a life table analysis in this regard would be more enlightening?

Kovar: I think that it might be helpful.

Epidemiology of Senile Dementia

Ernest M. Gruenberg, M.D., Dr. P.H.

Johns Hopkins University
School of Hygiene and Public Health
615 North Wolfe Street
Baltimore, MD 21205

Senile Dementia: A Disease

We often err in our thinking about "the aging process" as somehow synonymous with progressive deterioration in social and physical functioning. It is true that such deterioration is common among the elderly. But I am convinced that this is because senile dementia is such a common disease although, I concede to you, an incompletely understood one. Let me begin by describing some of the features of this common disease of old age.

Senile dementia is a clinically recognizable condition, with its own peculiar presenting picture. I quote from a recent article by V. A. Kral (*1*), "Senile dementia is a mental disorder of gradual onset and continual progression, characterized by a certain type of memory impairment—namely an amnestic syndrome, the essential feature of which is severe impairment of immediate recall, shortened retention span, disorientation, loss of recent and remote memories and sometimes confabulations." (Confabulation consists of making ready answers without regard to truth.) I quote from Kral, rather than from the DSM–II (*2*), or the WHO glossary (*3*), simply because he does a better job of describing the condition.

This clinical condition which the psychiatrist, family practitioner and internist frequently see is a manifestation of a specific brain disease known as senile brain disease. The lesions occur mainly in the hippocampus, fornix, and mammilary system of the brain. The pathology is visible on autopsy as senile plaques and neurofibrillary tangles which can be easily recognized by any trained pathologist.

The brain changes associated with senile dementia are somewhat perplexing. Statistically, there is a correlation between the quantity of brain pathology and the degree of demenita observed in cases who die. But some cases are far from the regression line. Indeed, at autopsy the brains of some people who had been extremely demented show virtually no brain damage, and individuals who were quite lucid during life have been found to have brains riddled with plaques.

There are other physical conditions which cause similar, or even indistinguishable, dementias—other chronic brain syndromes; e.g., cancer, arteriosclerosis, poor nutrition, chronic alcoholism.

The most common dementia is the senile dementia due to senile brain disease. It is so common, in fact, that one-fourth of the people in this room today can expect to develop it if they live to 80.

A word about Alzheimer's disease. Alzheimer's disease presents a pathological picture identical to that seen in senile brain disease, but it occurs before people reach the age of 65. It is an extremely rare condition, so rare that we are not exactly sure just how common it is—a generous estimate of its prevalence is less than 0.1 percent. But it is a favored research topic—probably because it hits people during their working years. In any case, Alzheimer's disease and senile brain disease may or may not be the same entity. If we saw a dip in the annual incidence rate curve for these conditions between ages 50 to 65, we could conclude that they are two different conditions and concentrate on the much commoner condition which causes senile dementia. But we should be concentrating on senile dementia anyway; because it is so common, it is much easier to study. What we need are clinical and pathological data on the same population, and the recognition that we are dealing with a disease, not a normal, inevitable aging process.

Epidemiology of Senile Dementia

We know painfully little about the epidemiology of this common crippler. In 1972, Kral (*1*) correctly reported that there were no epidemiological studies of the psychiatric morbidity of a truly representative sample of the aged population, so no true incidence and prevalence rates could be determined.

However, we now have some data which are currently in press (*4*) on a small (2,500 population) south Swedish population we call Lundby, which was screened for all psychiatric illnesses in 1947 by Essen-Möller and again in 1957 by Hagnell. Every member of the population, sick or well, was interviewed on both occasions, data were gathered for episodes in the intervening decade, and a great deal of care was taken to maintain consistent criteria for case identification. But when Professor Hagnell and I examined the data for cases of senile dementia—both psychotic and nonpsychotic—we discovered a curious phenomenon. The clinical syndrome of senile dementia had acquired a much longer average duration between 1947 and 1957. This finding emerged after several years of trying to unravel the mystery of why the total prevalence rate for all mental disorders in the Lundby population was significantly higher in 1957 than in 1947. By applying the 1947 age and sex-specific prevalence rates to each age and sex group in 1957, it was possible to show that much of the increase had occurred in the oldest age groups. Detailed examination of the data showed that up until about 1949, episodes of senile dementia lasted an average of less than 3 years and that episodes beginning after 1949 had much longer durations. Since this syndrome does not terminate except by death and the death certificates on the subjects were available through 1967, it was possible to perform the calculations which produced figure II–12. The population survivorship curve for the decade after 1947 parallels almost exactly the population survivorship curve for the decade following 1957, although the latter shows a slight improvement in expectation. When the cases of senile dementia present in 1947 are followed they die off quickly, but the more numerous cases in 1957 have a survivorship curve which shows a remarkable extension of life. This differential in the extension of life is attributed to the postponement of death from fatal intercurrent infections, particularly pneumonia.

The increased duration of senile dementia episodes in Lundby probably accounts for all of the increase we saw in its prevalence. The

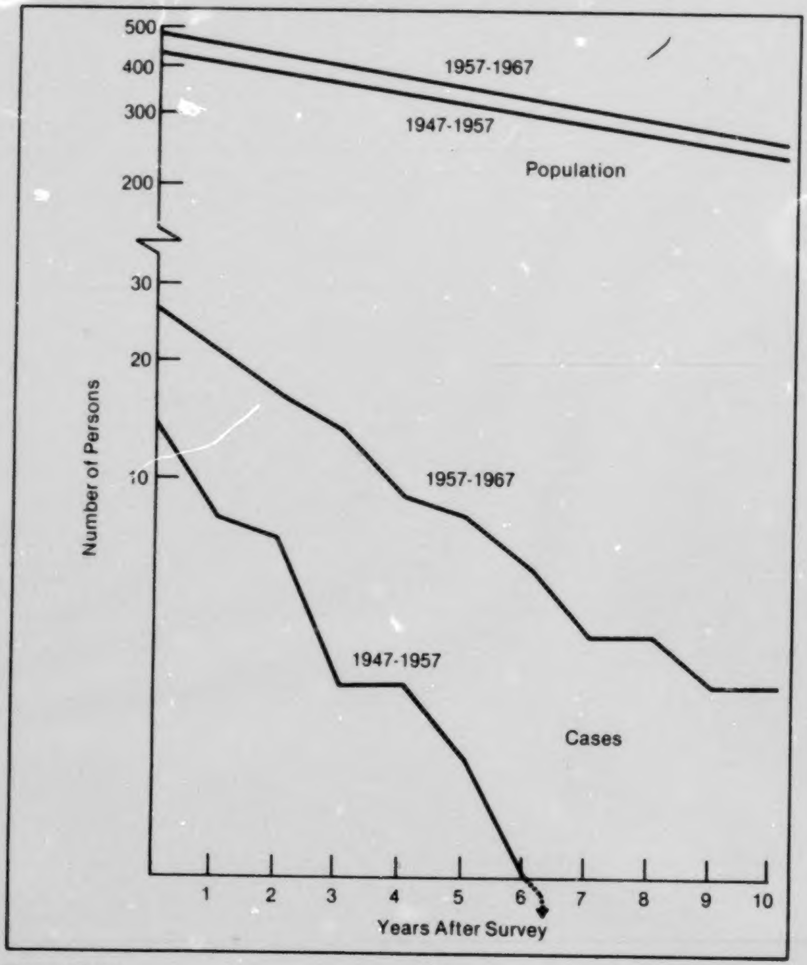

Figure II–12.
Survivorship curves for the general population and for senile dementia cases. Lundby, 1947–1957 and 1957–1967. (Reprinted with permission—see reference 4).

point prevalence rate for senile dementia in people over 60 on July 1, 1947 was 2.3 percent for men and 3.2 percent for women. In 1957 these figures had risen to 4.9 percent for men and 5.7 percent for women. Figure II–13 shows how the cases of senile dementia are increasing in the elderly population. Although there are too few cases to examine the

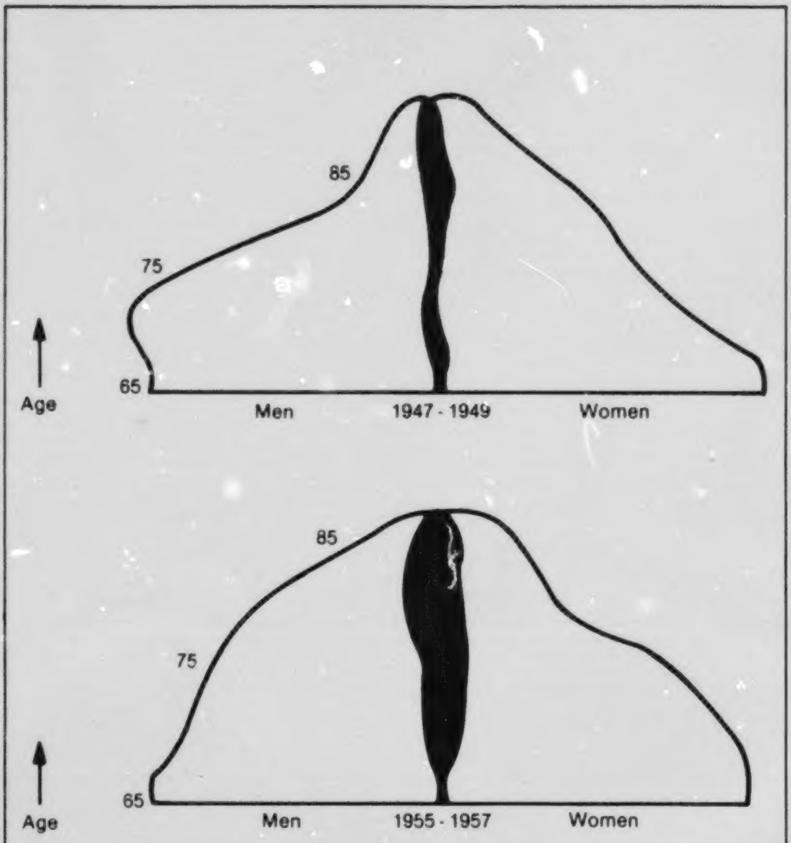

Figure II–13.
Age pyramid of the Lundby population over age 65 (Reprinted from reference *6*).

issue with precision, these rising prevalence rates do not differ significantly by sex or age group. It is surprising to be able to show such a big increase in the duration of illness episodes based on such a small population. But there is reason to believe that this phenomenon of rising prevalence rates of senile dementia is not unique to this population. The "friend of the aged," pneumonia, has been conquered, and is no longer able to carry off the old person in an "acute, short, not often painful illness" and prevent the "cold gradations of decay" so distressing to the individual and his friends (*5*).

A second important finding from the Lundby study is that the age-specific annual incidence of senile dementia rises with age until the early eighties and then seems to taper off or decline. Our population is too small to be certain that this is the case, although there is some good corroborating evidence. Tomlinson and Kitchener (*7*), working in Newcastle-upon-Tyne, found that the age-specific annual prevalence of the brain patnology also levels off in the ninth decade of life (figure II–14). This came from a consecutive series of general hospital autopsies. If it is true that the annual incidence rate of senile dementia levels off or declines during the eighties, this would have important implications for elderly people and for researchers as well. It would mean (if my calculations are correct) that about 30 percent of those who live past age 80 will never develop the disease We would then be able to conduct case-control studies on demented and healthy 90-year-olds, with confidence that an acceptably low percentage of our controls will later develop the condition.

Future Research

What are the next steps in epidemiological research? We have been asked to conclude our presentations by setting priorities for future research. This is not difficult for me to do. Data were gathered on the Lundby population in 1967 and again in 1972. Yet these data have not yet been analyzed. Neither Dr. Hagnell nor I can find funds to perform the analysis. These data would provide us with an additional 15 years of information on psychiatric morbidity in the same population about whom the decade 1947 to 1957 has already been studied. Thus a full quarter century of prospective data are there waiting for for analysis and interpretation. Despite the disadvantages inherent in using such a small population, this is the best data available in the world for certain epidemiological studies of senile dementia. Our first research priority should be to find out what these data can tell us.

From these data and from new studies we should try to determine with certainty whether the age-specific incidence of senile dementia does level or fall off after age 80. If so, case-control studies of senile dementia become feasible, beginning in the same population.

Whatever the answer to the incidence question, it is essential to have clinical and pathological studies on the same population. Tomlinson's autopsy data show that most cases of senile brain disease are "silent," i.e. produce no recognizable changes in mental functioning. In such future studies, it is important that interviewers be "blind" to the age of the people they are interviewing, so that preconceptions about losses of cognitive function due to "normal aging" do not contaminate the findings regarding losses due to disease.

Another intriguing question is the association between prior frequent infections and the probability of developing senile demenita. Is there an association between having senile dementia and suscepti-

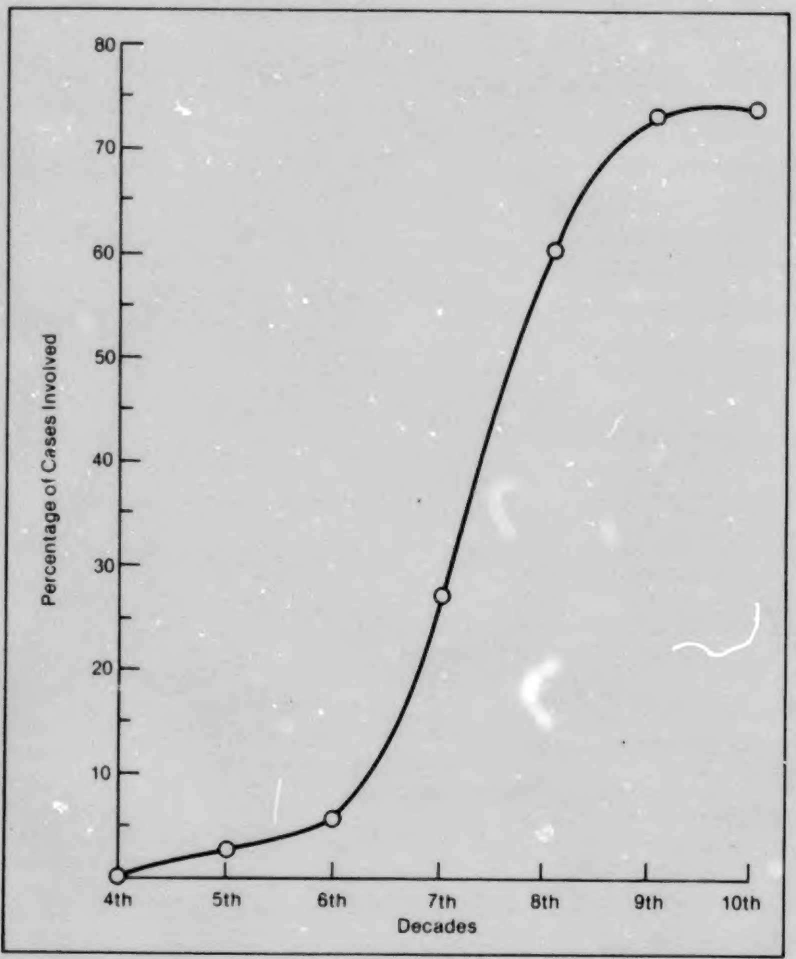

Figure II–14.
Percentage of cases involved with granulovacuolar degeneration in the fourth to the tenth decades in 219 routine hospital cases. (Reprinted with permission—see reference 7).

bility to infections? Despite the increased longevity of cases because of antimicrobial treatments, cases still tend to die of secondary infections, although at a later time. If the association with infections precedes onset of the condition, what are the possible causal implications? If it follows onset, what is the mechanism by which susceptibility to infection is increased?

Finally, we must ascertain what the precursors of senile dementia are. Because this condition is so common, we might phrase the question the other way around: What are the characteristics of the healthy 90-year-olds in the population? What keeps an individual from developing this frequent—but not ubiquitous—disease?

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Discussion

Waldron: With reference to senile dementia, you said that the incidence did not rise after age 80. But if it stayed constant, I do not see how that is the end of the age of risk.

Gruenberg: I do not either. I am simply saying that all I know from the epidemiological data is that it cannot rise. There are enough people above the age of 80 in the population that if the incidence were rising in a straight line, rising exponentially, or rising at all, we would expect many more cases in this age group than we actually see. The number of people is too small however, to decide whether the incidence rate is falling or just staying steady. I have no idea what the curve looks like after age 80.

The Lundby population experienced 2,500 person-years of exposure in that first study, and this figure will more than double when we add another 15 years of data. Besides that, the aging of the population will throw more people into the age group at risk. As time goes on, more people are living longer, so we will have to figure them in. (Preliminary analysis of the 15-year data now reveals that incidence continues to rise with age in this population, i.e., we do not see an end to the age of risk.)

Brody: Was there any sex difference in either the clinical or pathological features?

Gruenberg: Yes, there is a difference, but not a statistically significant one. The n's are small but there is a slight tendency for the condition to be more common in women by age 80. How much of that is due to longevity in general, I do not know. But that is why I gave you one chart for the whole. In each age group one sex differs a little bit from the other, and from the total population, but none of these differences is statistically significant within a given decade of observation.

Brody: I raised the question because of our studies in Guam. There is a disease we call Parkinson's dementia. One in 10 adult Guamanians dies from it. It disappears by about age 70. There are no senile plaques or very rarely are senile plaques seen in this disorder. It is essentially neurofibrillary degeneration.

Guamanians over about age 40 start accumulating neurofibrillary degenerations at a high rate. This accumulation goes on with age as far as we can see from the control brains we collect and is equal by sex. However, the clinical disease that is registered shows a three to one male excess.

Gruenberg: You are telling me something interesting which I am glad to hear. It is an additional piece of information, but I do not know how to connect it. You know you cannot get very excited about the sex distribution of the data from Newcastle. It is just a beginning. The Newcastle survey is the best body of data I have been able to find in the literature. I have had no contact with the director myself. But the data clearly indicate to me that there is a need for such surveys. Hopefully, if populations are to be examined for this disorder, they will be defined populations. The Newcastle population is not defined; it is from general hospitals; they find it at autopsies.

Kannel: It seems to me you have a similar situation to coronary disease. If you look at postmortem data, you find almost everybody has some degree of coronary artery disease. Yet, if you look at the incidence you note a wide range in the rates for clinical events. The question then comes up (as in coronary disease), both in terms of coexisting pathology and in terms of predictions, as to how good the correlation is between the number of plaques and the severity of dementia during life.

Gruenberg: I can only answer that on two grounds. I know that Martin Roth is very convinced of a high correlation between clinical dementia and the number of plaques in particular parts of the brain. But I also think it is very clear that at the present time pathologists have much better measures of coronary disease than of senile brain disease. As far as I can make out, neuropathologists have not agreed on techniques and sets of standards, and so cannot say anything consistently from one study to another as to how much senile plaque formation there is in the brain.

I did a review of this topic. There are several pathologists working on the question and each one is advocating a different set of criteria for severity. Within this group, you can analyze the data for some idea of consistency. But apparently, there is a great deal of difference in how you sample the cortex and how you count the plaques. Procedures seem to be just approaching standardization, so far as I can see.

Kannel: Is there any relationship between dementia and the degree of atherosclerosis in the cerebral circulation?

Gruenberg: Apparently not. I would summarize the situation by saying that the evidence that atherosclerosis and senile brain disease have a common cause is about as good as saying athletes' foot and dandruff have a common cause based on the fact that you have skin. Here you have brain, and the artery disease will interfere with the circulation of the brain and lead to atrophy of the brain. There certainly are cases that have both senile dementia and atherosclerosis; there are cases that have only the one, there are cases that have only the other. Both are very common conditions and as far as I can make out from the available data, the association of the two is just about what you would expect from the prevalence of each.

Kannel: Do you think the clinical criteria you cite are acceptable and do they distinguish this dementia from other dementias and from things like depression?

Gruenberg: I think the question you are getting at really, is are they distinguishable on examination. In the first study I did in this field quite a while ago, I created an entity called cerebral arteriosclerosis and senile psychosis. I felt that anyone who thinks he can tell them apart clincally is foolish—I just counted them as a combined entity.

But the fact is that today I am quite convinced we can do much better than that. There have been plenty of studies back and forth from the pathology table to the clinicians, starting with studies just after the war. It is getting clearer how to tell the two conditions apart. Still, at the clinical level, you are going to find people whom we have reason to suspect have both. If you want to predict what the brain is going to look like or the degree of dementia, it does not matter how much evidence there is of arteriosclerosis. The more dementia there is, the more likely you are to find senile plaques, even if there is arteriosclerotic brain disease too. Now in lots of arteriosclerotic brain disease there is obviously no plaque formation.

Ostfeld: In the citation you made of the annual incidence of 5 percent for Alzheimer's disease in the very late years of life, what were the criteria for determining an incident case?

Gruenberg: The description that I quoted from Kral is the closest description in English that I have found. Of course, the Lundby investigators are talking Swedish when they distinguish these cases, and I have been over their criteria with them very carefully. But the 5 percent is not confined to the psychotic cases—the cases who have active disorientation, need a lot of personal care and that sort of thing. The amnestic syndrome and the absence sometimes of signs associated with arteriosclerotic brain disease or tumors or depression or something of that kind distinguish senile dementia. The nature of the amnestic syndrome at interview and information from other members of the same household and the family physician as well indicate that the commonest early sign is an observable loss of retention and recall. I do not know how much you want me to elaborate, but I want to be sure that you understand that I am not talking only about psychotic senile dementia. I am also talking about those who were not psychotic.

Palmore: That was prevalence.

Gruenberg: That was prevalence. That is the way it started. That is how the cohort began. Then, 10 years later, over a 3-year period, Hagnell went back to the same population wherever they were. He interviewed everybody who was still alive, and took a complete psychiatric and medical history of the intervening decade. He examined and personally interviewed around 98, 99 percent of the population that was still alive. I did not report any of the episodes that started in the intervening decade. I gave you two point prevalence rates. Hagnell has the data on episodes of the same conditions that started during the decade. It is from those pieces of information that I am able to make an estimate that is in the neighborhood of 5 percent per year.

Ostfeld: I think that is a very important piece of information because even if the annual incidence is 2 or 3 percent instead of 5 percent, a prospective study is feasible.

Gruenberg: I quite agree with you and I am convinced of his retrospective diagnosis. He is a conservative man and he was not going to make a diagnosis on the basis of partial information. Most of the cases in which he made a diagnosis were still alive and were ready to be examined by him directly in 1957. Then he had to interview the family and everybody else to assign the year of onset.

Ostfeld: There are two kinds of methods of making diagnoses that I have considered and both were unsatisfactory for different reasons. One is by defining a case as a certain percent decrement on some test of memory or cognition; this has so many errors and problems with it that I do not buy it. The other one is saying the fundamental loss is a loss of a capacity to direct the activities of your own life. That is, when somebody has got to tell you to get up, when to dress, when to eat, then you have got dementia. If you limit it to the second definition, it has an extremely low incidence. So, I have been whip-sawed between two unsatisfactory definitions. If a clinical interview can demonstrate a condition with an incidence of even 2 or 3 percent, then I think you can do a prospective study and that is a very important piece of information.

Gruenberg: I quite agree with you. I would like to say, parenthetically, to help you have some confidence in these data, that I met Dr. Hagnell in 1959. I went to visit Essen-Möller. Hagnell was going to go out interviewing and he invited me to go with him, but he said I would be bored unless I understood Swedish. I said I would not be bored and went to observe the silent interviewing. But I had been impressed previously by Essen-Möller's data. Essen-Möller's primary interest is not mental disorders but personality traits and he has classified each person on five personality dimensions. In order to do that you have to have a fairly long interview with each person. They picked up this information about illness incidentally.

These interviews took 45 to 50 minutes each, in a very relaxed, come back tomorrow and finish sort of mood. The way the whole thing started was through their interest in the genetics of personality traits. That made me have more confidence in their description of a clinical syndrome. They had no basis for personality traits except the face to face interview.

Ostfeld: I do not knock period prevalence data at all. We made an estimate of incidence based on period prevalence. We estimated that the incidence of stroke in a defined population in Chicago would be 2 percent per year and then we did a 5-year cohort study of 4,800 people and came up with an annual incidence of stroke of 2 percent per year.

Gruenberg: This is not a period prevalence. This is the incidence data I gave you for the decade 1947 to 1957. It was historically collected data about the episodes which began during that decade. It is prospective data retrospectively gathered—nonconcurrent prospective data.

Speaker: Can you pick up these plaques with CAT scanning techniques?

Gruenberg: I have been trying to find out whether CAT scan techniques will pick up senile plaque formations and slight degrees of atrophy. I would like to know the answer to that. I think there is also another clue pointing in the same direction, that is, pathologists are describing little deposits of aluminum in these neurofibrillar tangles. It seems to me that it may be possible through psychological and cellular devices to identify that with noninvasive techniques. Brains in life would have these deposits. What you are looking for then is a noninvasive technique for examining these brains. That is what I am looking for too. If we had that it would change our whole research strategy.

Sacher: What are your thoughts on the etiology of the disease?

Gruenberg: Well, that is the amazing thing about the disease. There are no data to base any theories on about etiology. We have only the most elementary base of information. We do not know the difference between people who get it and people who do not get it. But people who get it live longer.

Sacher: How about a genetic basis?

Gruenberg: There is some recorded evidence. Kallman has presented some evidence which suggests that senile dementia is more likely in identical twins But this is all very closely related to survivorship. It is very hard to do the relevant kind of familial aggregation study unless everyone survives for a long time, which they refuse to do for you. When you have a condition with such a high incidence rate, it is hard to tell. However, one thing I will point out is that the younger age group has a much lower incidence of Alzheimer's disease; so 0.1 percent could be a high death rate. There are no data on the incidence of this condition in the forties, fifties and into the sixties from anybody that I could find. I do not see any point in concentrating on those young cases for epidemiological research when you have such high annual incidence rates in older age groups for study. But I do not know anything about the etiology. My ideas are no better than yours. I would like to get some data before I started speculating.

Sacher: Has any association with a past history of infectious disease been observed?

Gruenberg: One of the interesting things about senile dementia is its association with death from pneumonia. Whether this association occurs because infectious diseases make people more susceptible to senile brain disease and senile dementia, or because senile dementia and senile brain disease change the resistance of the organism and make people susceptible to pneumonia is a moot question. However, if we could find good patient controls in this body of data we could look at that question prospectively over 25 years. We know who reported in 1947. We could find out who had frequent infections; Essen-Möller was interested in psychosomatic illness, so he recorded that. If we could find controls for these people we could look at the question of risks and longevity.

Kannel: This issue of adding a lot of pathology and very little clinically recognizable disease suggests either that there is some critical amount of disease necessary to produce recognizable clinical deficiencies, or that perhaps you should be looking at the rate of fall off in intellectual function in relation to various hypothesized risk attributes. Therefore you ought to do tests of intellectual function of various sorts; follow these persons and determine how they fall off, who has a rapid fall off and whether there are quantitative changes. Like coronary disease, there may be a critical value. You do not get much in the way of myocardial damage or symptoms until you get very severe stenosis of the coronary artery.

Gruenberg: In my short paper I did not bother to summarize the research on that question. But Martin Roth and his associates discuss it. If I understand it correctly, it is in the hippocampus. By their methods, if you find more than nine plaques per field in the microscope you are going to have evidence of senile dementia with a high probability. If you find less than nine plaques per field in the hippocampus, then you are not going to have senile dementia. So they had a combination of threshold and a particular location.

Costa: The little reading I have done in this area, you could say including Shelanski and Poskanzer in the previous conference, would indicate a very mysterious, if any, correlation between the vascular or cellular structure of the cortex and subcortex and mental performance. Can you give me a citation that indicates this greater specificity of clinical and pathological findings?

Gruenberg: The studies of Roth, Tomlinson and their group in Newcastle and the studies at Lundby are the two major ones. The particular curve that I showed you (figure II–14) is an unselected case, but in the same set of articles by Tomlinson and associates, there are similar data about people who were given good mental status examination during their terminal illness. One of the main things about the problem today is that there is only one group in modern times that has dealt with this interrelationship and all of these people have been associated with Martin Roth. I think he deserves a lot of credit for it, but until others start looking for the same kind of phenomena, the guy at Green's Square has an entirely different way of measuring both psychopathology and brain pathology.

Costa: One final question. Can you predict the mental decrement from the cellular changes? I know that where you find the dementia, more often than not you find some accompanying degeneration.

Gruenberg: That is what I think Martin Roth is saying, that if one sees more than nine plaques per field, in that particular area of the brain, it is almost certain that the person has dementia, by his criteria. Now one of the difficulties there (and I want to go see him about it) is not only a problem with the pathologist, it is a problem with the clinician too. I cannot quite figure out exactly what clinical criteria Roth is using from his multitude of publications. He has obviously learned from experience as we all do and I am not sure which criteria he was using in those last studies. I would like to know more about it.

Schneider: I am glad that Dr. Sacher brought up the point of etiology because I think that once you consider this very strongly, and in terms of etiology, there is increasing evidence over the last few years for a slow viral etiology for a number of neurological disorders.

Gruenberg: You can have it. You can have genes; you can have slow viruses; you can have social stress; or you can have survival of the unfittest because of the lowering of the mortality rate. There are a number of hypotheses that are perfectly credible. That is why I did not pick any, because they are all equally credible to me. You can reach way, way out: the evidence is so lacking that if you said that it was because they were spanked when they were children, I could not disprove it.

Schneider: That is not what I am talking about.

Wilkie: Dr. Sacher was talking about the incidence of infection and now Dr. Schneider is talking about slow viruses. Buckley has one study out and I think Cohen has one coming out in *Science* shortly on alterations in the immune system in patients with defined Alzheimer's disease. I believe it is IgM and these are in turn related to performance in a number of behavioral tasks.

Gruenberg: The breakthrough may come that way through straight laboratory clinical studies. It is possible as far as I can estimate the situation today. The answer may come from way out in the corner someplace that we are not anticipating. That is one way and I think it certainly worth looking for. I will not predict where the new knowledge is going to come from.

Blood Pressure and Cognitive Functioning

Frances L. Wilkie, M.A.

Department of Psychiatry and Behavioral Sciences
School of Medicine
University of Washington
Seattle, WA 98195

It is estimated that 23 million Americans have hypertension (*1, 2*), a chronic disease which increases in incidence with age and is frequently accompanied by atherosclerosis, heart disease, strokes or kidney impairment (*3, 4, 5, 6*). Relatively little attention, however, has been paid to the consequences of hypertension on cognitive functioning.

A comprehensive review of the literature relating performance to hypertension has been accomplished by Spieth (*7*), Abrahams (*8*), and Eisdorfer and Wilkie (*9*). Topics have been selected for inclusion here which illustrate the potential for modifying the relationship between blood pressure (BP) and cognitive functioning, as well as to point out the complexities involved. Specifically, discussion will focus upon the effects upon cognitive functioning of antihypertensive drugs and other techniques used to lower BP, differential rates of decline in performance as a function of BP level, and possible underlying mechanisms accounting for the relationship between performance and BP.

The relationship between BP and cognitive functioning is a complex one. To date, the findings have been correlational in nature and relatively little is known about the underlying physiological and psychological mechanisms (*9*). Numerous investigators (*7, 8, 9*) have found, however, that middle-aged and elderly hypertensives perform less well than age-matched normotensives on a variety of tasks measuring intellectual functioning, organic brain impairment, and psychomotor response speed.

Antihypertensive Medication, Biofeedback Training, and Performance

Evidence indicates that the successful treatment of hypertension prolongs life and that morbidity and mortality from strokes, congestive heart failure and renal failure is delayed and drastically reduced (*10*). Based upon these findings, one would expect that the incidence of senile dementia associated with vascular alterations would undoubtedly be reduced when hypertension is treated (*11*).

It is important to note, however, that the acute as well as the chronic effects of antihypertensive drugs upon cognitive functioning have not been systematically examined among middle-aged and elderly hypertensives. Since there are three general classes of antihypertensive agents—diuretics, adrenergic nervous system inhibitors, and vasodilators—one could speculate that there would be differential drug effects upon performance depending upon the mechanism of action, dosage level, and the patient's tolerance of the drug.

Unfortunately, the effects of antihypertensive drugs upon performance among hypertensives has not been examined within the framework of pharmacologic mechanisms. Instead, most studies were conducted in the context of a specific problem (e.g., Do hypertensives perform less well than normotensives? Are there differences in performance between treated and untreated hypertensives?).

Chronic effects.—In a review of his work, Spieth (7) reported on findings which tend to suggest that antihypertensive drugs (vasodilators) may enable hypertensives to maintain their level of cognitive functioning at least as well as their normotensive age peers. He examined the relationship between cardiovascular status and performance on a variety of tasks among men aged 35 to 59 years of age. Most of the men were pilots or air traffic controllers undergoing medical certification by the Federal Aviation Administration. For comparison purposes, subjects were also included who were receiving treatment for hypertension and/or coronary heart disease. A battery of psychological tests was administered. The subjects were informed that the psychological tests were for research purposes and had nothing to do with the certification process.

When compared with the healthy group, Spieth found that subjects with arteriosclerotic heart disease were significantly slower, independent of whether they were being considered for certification or not. A normotensive group, presumably under stress since they had been falsely classified as having heart disease, performed about like the healthy group, as did another group with evidence of mild to moderate congenital or rheumatic heart disease.

Interestingly, the hypertensives whose BP was maintained within normotensive limits by antihypertensive medication (and not being considered for certification) performed about like the healthy group. In contrast, the untreated hypertensives (being considered for certification) performed about like a group with cerebrovascular disease, with the latter group having the slowest response speeds of all groups.

As pointed out by Spieth, it is difficult to interpret the effects of antihypertensive medication upon performance from his data because situational stress and hypotensive drugs were confounded. This would be true if the assumption were made that the subjects undergoing certification did experience stress during the psychological testing, although it was independent of the certification decision. As noted earlier, however, other groups either with or suspected of having cardiovascular disease were able to perform as rapidly as the healthy group although they were being considered for certification. Thus, it is unclear from Spieth's findings to what extent, if any, situational stress may have differentially affected the performance of the treated and untreated hypertensives. Nevertheless, Spieth's findings on the effects of antihypertensive drugs and stress upon performance are interesting. It is surprising his line of research has not been followed.

Acute effects.—Light (*12*) reported on the acute effects of lowering BP upon psychomotor response speed using a serial reaction time task similar to that employed by Spieth (7). Her study included normotensive as well as previously treated and untreated hypertensive men and women aged 18 to 59 years of age. Hypertension was defined as a BP of 140/90 mm Hg or greater. Subjects were judged free of coronary heart disease, signs of congestive heart failure, or cerebrovascular disorders. The treated hypertensives were taken off drugs from 3 to 21 days prior to the administration of a powerful diuretic (furosemide) to lower BP in order to determine their plasma renin (PRA) levels. The reaction time task was given about 2 hours after the drug was administered.

Light's findings were rather complex. When PRA was not considered and the age effect was statistically controlled, the previously treated hypertensives had slower response speeds than their untreated counterparts, with both hypertensive groups slower than the normotensives. When PRA was considered, the slowest response speeds were found among the untreated hypertensives with high PRA. At normal and low PRA levels, the treated hypertensives were slower than their untreated counterparts. Light interpreted her findings on response speed in terms of etiology and severity of the hypertensive disorder as well as in terms of the acute effects of lowering BP upon cerebral blood flow.

In some cases, high PRA levels among hypertensives may be indicative of renal arterial insufficiency which has an acute and symptomatic onset and is generally diagnosed and treated at an early stage. This may, in part, have accounted for the poorer performance found among the untreated hypertensives with high PRA. Low and normal PRA levels appear to be related to hypertension involving a different etiology (than high PRA), with a much slower progress associated with a greater risk of developing arteriosclerotic changes. Thus, individuals with low or normal PRA would be less likely to receive the treatment during the early, more labile, stage of hypertension which would allow the disease to run its course. As noted by Chrysant and colleagues (*1*), hypertension tends to accelerate the arteriosclerotic process, which in turn aggravates the hypertension—and a vicious cycle has begun.

Although Light (*12*) did not measure cerebral blood flow in her subjects, she postulated that the acute effects of lowering BP may have led to difficulties in adjusting to the lower BP in some hypertensives. This may have resulted in reduced cerebral blood flow and ischemia which in turn was accompanied by an inability to respond quickly. She supported this contention by noting Bentson and colleagues' (*13*) findings that cerebral circulation was reduced in both treated and untreated hypertensives when their mean arterial pressure fell to about 95 mm Hg.

It is difficult to interpret the acute effects of antihypertensive drugs upon performance from Light's (*12*) data. One serious problem was that response speeds were measured *after* but not *before* the diuretic was given. In addition, the effects of antihypertensive drug cessation may have confounded her findings, since the treated hypertensives were taken off their regular medications from 3 to 21 days prior to testing. It is also not known what effect drug cessation may have had upon renin levels or how the drug, furosemide, may have interacted with any of these variables.

In view of Light's finding that the slowest reaction times were associated with high PRA, it is interesting to note that Campese and colleagues (*14*, *15*) have found that plasma catecholamine (CA) levels were related to plasma PRA levels. The CA neurotransmitter, norepinephrine, is a potent pressor agent which is also highly responsive to stressful or noxious stimuli (*16*). In Campese's study (*14*), 70 percent of the hypertensives with high PRA had higher CA levels than their counterparts with low or normal PRA. As a group, the hypertensives had 27 percent higher CA levels than the normotensives, with this difference, of course, greater if the comparison involved only the hypertensives with high PRA. They also found that the antihypertensive drug, propranolol, a beta-adrenergic blocking agent was most effective in lowering BP among the hypertensives with high CA and PRA levels. They suggested that high PRA in primary hypertension may be an expression of sympathetic hyperactivity and that the antihypertensive activity and the PRA lowering effects of propranolol were possibly related to an inhibitory effect on the sympathetic nervous system.

As discussed by Hoeldtke (*16*) there has been considerable speculation that hypertension is a disease of noradrenergic neurons, since norepinephrine (NE) is a potent pressor agent. Alterations in NE metabolism have been observed in a variety of animal models of hypertension as well as in animals made hypertensive by drugs. Much of the early work in this area, however, was based upon urinary catecholamine levels with the findings often contradictory. The recent development of assays for plasma catecholamines, along with assays for plasma renin and angiotensin, should shed further light on this controversial area. As noted by Hoeldtke, recent findings indicate that plasma NE levels of hypertensives are positively correlated with diastolic BP. In view of the variability in the circulating NE in hypertensives, it is important that future studies of catecholamine metabolism in hypertension characterize the renin-angiotensin system and segregate that group of individuals with decreased sympathetic neuronal activity and PRA responses. Since the catecholamines are highly responsive to physical as well as psychological stressors (*16*, *17*), these factors should also be considered in studies of hypertension and performance.

Although it is not clear whether the effects of antihypertensive drugs upon the performance of normotensives can be generalized to a hypertensive population, nevertheless, much of the research on the effects of these drugs on performance has been based upon young normotensive subjects. For example, there is a growing but contradictory body of literature on the effects of propranolol on the performance of normotensive young individuals. It is somewhat difficult to interpret the findings due to differences in dosage levels, varied time intervals between drug taking and psychological testing, and the use of a variety of behavioral measures. Bryan and colleagues (*18*), using a single oral dose of propranolol, found decreased heart rate accompanied by an increase in simple visual reaction times, and a performance decrement in complex hand-eye coordination. No significant changes were found in various ophthalmological measures. No decrement in performance was observed in several studies using a single oral dose of 40 (*19*) or 120 mg (*20, 21*) of propranolol, although there was increased variability in performance at the lower dosage level. Ogle and colleagues (*22*) found that 240 but not 320 mg of propranolol impaired pursuit rotor performance, but the performance measures were obtained at different time intervals following the administration of two dosage levels.

Although the neuropathologic data indicates that propranolol crosses the blood brain barrier and thus has a central effect in both animals and man (*23*), several studies have reported no central effect in terms of behavior (*20, 24*). In contrast, Straumanis and Shagass (*25*) found that propranolol eliminated alterations in somatosensory evoked response (SER) amplitude which were due to the drug, triiodothyronine (T3) which reportedly increases the excitability of neurons in the spinal cord, cortex, and hypothalamus (*26*). In the absence of T3 produced CNS excitability, propranolol had no significant effect on SER and EEG measures among their young subjects (*25*).

Since propranolol has been found effective in lowering anxiety levels (*27–34*), the drug's effect upon performance may, in part, depend upon the subject's physiological arousal level before the drug is administered. Since there appears to be an optimal level of arousal that is good for performance, while hypo- or hyperarousal may be detrimental to performance (*9*), propranolol may either be beneficial or detrimental to performance depending upon the individual's state of arousal and the extent to which it is modified by the drug.

There is some indication that the older individual may experience greater stress in the psychological testing situation than the young (*34, 35, 36*). Eisdorfer (*36*) has proposed that the deficit observed in verbal learning performance among healthy old men is associated with heightened arousal as measured by autonomic nervous system activity. In an attempt to improve performance by lowering arousal levels, Eisdorfer, Nowlin, and Wilkie (*34*) administered propranolol (10 mg. IV) to a group of healthy old men while another group received a placebo. The group receiving propranolol had a decrease in heart rate and free fatty acids, accompanied by significantly better performance than was found among the placebo group. Although supporting data on animals have been reported (*37*), some doubts have been raised as to the generality of these findings in studies which have typically used lower dosage levels under low stress testing conditions (*38*). This line of research has not been followed with the hypertensive elderly.

Side effects.—As discussed by Nies (*39*), a lack of compliance with the therapeutic regimen is a major problem in the management of hypertensive patients. This lack of compliance is frequently due to side effects of the antihypertensive drugs, which is generally due to the pharmacologic actions of the drugs. There have been numerous clinical reports that the antihypertensive drugs, especially those which act upon the central nervous system, may produce sedation, inability to concentrate, depression, and diminished mental acuity. These effects may be transitory in nature and often can be eliminated through careful control of dosage levels, the combinations of antihypertensive drugs, or by switching to other hypotensive drugs with a different mechanism of action (*39*, *40*). This area should be studied further, particularly among the aged who may be more responsive to drugs and may already have compromised cognitive functioning.

Although it is known that antihypertensive drugs may reduce the hypertensive individual's risk of developing serious cardiovascular complications, comparatively little is known about the effects of these drugs upon cognitive functioning. One could speculate that the degree to which hypotensive drugs may affect performance will, in part, depend upon their mechanism of action, the degree to which adrenergic activity has been dampened, the severity of the disease process, and the individual's age. There is a need to examine the acute as well as the chronic effects of antihypertensive medications upon cognitive functioning, as well as to explore alternatives to drug usage as a means of lowering BP and how this may relate to performance.

Biofeedback training.—Goldman and colleagues (*41*, *42*, *43*) in a series of studies reported that BP was related to performance on the category subtest of the Halstead-Reitan neuropsychological battery. In two studies (*42*, *43*) these investigators reported that biofeedback training was successfully used to lower BP among middle-aged hypertensives and was accompanied by improvement in performance on the category test. Defining hypertension as a BP of 140/95 mm Hg, they found that biofeedback training resulted in an average reduction in systolic and diastolic pressures of 5 and 15 mm Hg, respectively, with the greatest reductions in pressure significantly associated with the greatest improvement in performance. A control group whose BP was monitored but received no feedback training showed no significant change in BP and no improvement in performance (*42*). In a subsequent study (*43*), these investigators replicated their earlier findings and also reported that the initial BP levels were significantly correlated with a life-stress questionnaire rating. Thus, the hypertensives who reported the most life stress had the highest BP levels, but the stress measures were not related to ability to lower BP during training.

Performance With Borderline High and High Blood Pressure

It is important to note that the findings on the effects of antihypertensive drugs upon performance were primarily based upon studies of individuals under the age of 60 years. It may, however, not be appropriate to generalize findings on the effects of antihypertensive drugs on cognitive functioning among the young and middle aged to the elderly. Indeed, there has been some speculation that mild elevations of BP may be necessary for the maintenance of cognitive functioning among some aged individuals (*44, 45, 46*).

Although drug usage was not controlled, Wilkie and Eisdorfer, in a series of reports (*47, 48, 49*) presented longitudinal findings on changes in performance according to BP levels among the aged. The subjects were men and women initially aged 60 to 79 years of age, who were community volunteers in a study of aging conducted by the Duke Center for the Study of Aging and Human Development. The project was not designed specifically to investigate the effects of BP upon cognitive functioning, but rather the BP measures were obtained during the routine physical examination.

Diastolic rather than systolic pressure was used in grouping the subjects, since the two measures were significantly correlated and diastolic BP was believed to be less sensitive to minor fluctuations. Approximating Masters and colleagues (*50*) classification, subjects were divided into a normal group with pressures between 66 and 95 mm Hg; a borderline high group with pressures between 96 and 105 mm Hg; and a high group with pressures greater than 105 mm Hg. The BP measures were obtained at the initial evaluation. Compared with the normal and borderline high groups, the high BP group had significantly greater evidence of end-organ change (that is, a cardiac-thoracic ratio of greater than 50 percent; eyeground changes of grade II or III; and left ventricular hypertrophy). Because of the nature of the longitudinal study (several years separated the evaluations), drug usage could not be controlled.

Simple and choice reaction time measures were examined as a function of BP among these elderly subjects who completed a 10-year followup study (*47*). None of the hypertensives initially aged 70 to 79 completed the followup study. At the initial evaluation, BP was not related to response speed. At the followup evaluation, the hypertensives initially aged 60 to 69 showed a marked slowing in response speeds while their age peers with normal or borderline elevations showed little change. Among those initially aged 70 to 79, the borderline high group had a slowing in response speed, while their age peers with normal pressures showed little change.

Among these same subjects, Wilkie and Eisdorfer (*48*) examined intellectual functioning as measured by the Wechsler Adult Intelligence Scale (WAIS) as a function of BP. Changes in the WAIS scores over the 10-year followup period were examined. In addition, the initial WAIS scores of the returning subjects were compared with those of their counterparts who did not complete the followup study.

Changes in the WAIS scores (as delta scores) between tests one and four were examined as a function of BP, with the results indicating that there were significant differences between the 60 to 69 but not 70 to 79-year-old BP groups on the Full Scale and Performance Weighted (Wt.) delta scores. As noted earlier, none of the 70 to 79-

year-old hypertensives completed the followup study. The changes in the WAIS Performance Wt. scores over the 10-year followup period are shown in figure II–15. Among those initially aged 60 to 69, the group with normal pressure remained relatively stable, while the high BP group experienced a significant loss. In contrast, the borderline high group showed a significant increase in performance, which was interpreted not as an increase in intellectual functioning but rather that they probably remained relatively stable but benefited from the

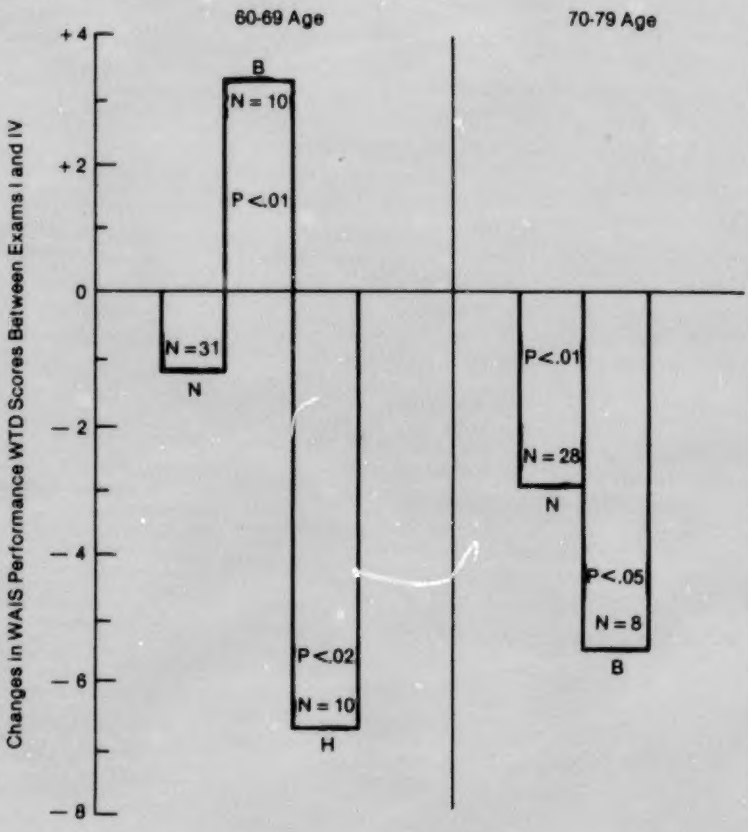

Figure II–15.
Intellectual change (delta scores) over a 10-year period, as measured by the WAIS, among individuals initially examined at ages 60 to 69 and 70 to 79 with either normal (N), borderline elevated (B), or heightened (H) diastolic blood pressure on the initial examination. (From reference *48*).

practice of taking the test four times during the followup period. This finding among the borderline high group tends to support Obrist's (*44*, *45*) contention that mild elevations of BP may be necessary among some aged in order to maintain adequate cerebral circulation. That this may not be true at all ages, however, is seen among those initially aged 70 to 79, where the borderline high as well as the normal pressure group experienced a loss in performance over the 10-year period.

The effects of age and BP upon intellectual functioning were also found at the initial evaluation, when the subjects who returned to complete the followup study (groups R) were compared with their counterparts who did not return (groups NR). The WAIS Full Scale Wt. scores for the R and NR groups at each age (60 to 69 and 70 to 79) and BP levels are shown in figure II–16. Among those aged 60 to 69, the returnees (R) as a group were superior to the nonreturnees (NR) as a group. Among the subgroups, only the returning high BP group was significantly superior to their nonreturning counterparts. Although the WAIS scores were not related to BP among the 60 to 69 year-old returnees at the initial evaluation, the nonreturning hypertensives had the lowest score of all groups. Among those initially aged 70 to 79, BP was significantly related to the WAIS scores, with the hypertensives having the lowest scores. At the older age, BP was significantly correlated with 12 of the 14 WAIS scores at the initial evaluation.

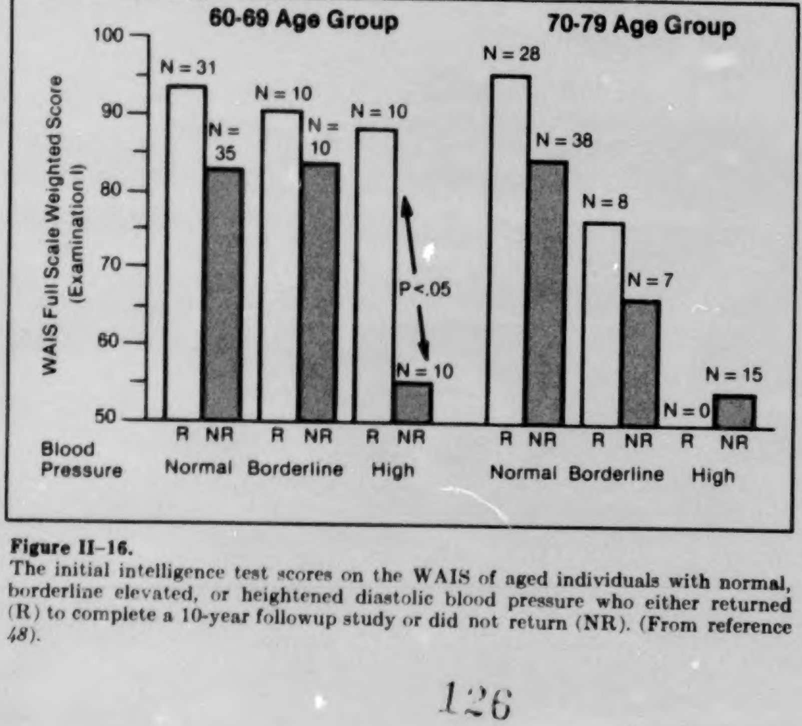

Figure II–16.
The initial intelligence test scores on the WAIS of aged individuals with normal, borderline elevated, or heightened diastolic blood pressure who either returned (R) to complete a 10-year followup study or did not return (NR). (From reference *48*).

Among these returning 60 to 69-year-olds, memory loss as measured by the Wechsler Memory Scale was examined as a function of BP (*49*). As seen in figure II–17, BP was not related to memory scores on the initial evaulation. At followup, however, the high BP group showed a slight decline on all tasks, with a significant difference found on the visual reproduction task, which involves a memory as well as a psychomotor component. Although not shown here, the high BP group's poor performance at the followup evaluation was found only on specific subtask items on both the visual reproduction and logical memory tasks. This finding did not appear to be related to item difficulty alone and suggested that performance factors rather than an inability to remember was involved. The performance of the borderline high BP group was about the same as that of the normal pressure group.

At the initial evaluation of the subjects participating in the Duke longitudinal study, Busse and Wang (*46*) examined the relationship between BP and EEG activity. They found a slowing in EEG frequencies when mean arterial pressures were below 95 and above 105 mm Hg, but not at pressures between these limits. They suggested that their findings confirmed the observation of an association between severe hypertension and cerebral complications. On the other hand, they suggested that mild elevations of BP may be necessary in some aged individuals with cerebral atherosclerosis in order to maintain an adequate blood supply to the brain.

Little attention has been paid to the effects of antihypertensive drugs upon the performance of elderly individuals with mild and high elevations of BP. If hypotensive drugs should be accompanied by reductions in cerebral blood flow, then one would expect performance to be adversely affected. In view of the controversy as to whether hypertension should be treated in the elderly (*51*) and the extensive use of a variety of drugs that can cause hypotension, there is a need to pay greater attention to the effects of drugs upon the performance of the aged.

Performance Decrements: Underlying Mechanisms

"**Cerebral blood flow.**—Since hypertension may be accompanied by atherosclerosis, heart disease, and strokes, cerebral circulatory insufficiency has long been suspected as the major underlying mechanism accounting for the performance decrements observed among hypertensives. Under normal conditions, mechanisms of autoregulation ensure a remarkably stable blood flow to the brain over a wide range of pressures, so that a marked drop in blood pressure must occur before cerebral blood flow is reduced. As discussed by Obrist (*45*), individuals with hypertension and elevated cerebral vascular resistance may be more sensitive to pressure changes which can lead to altered cerebral blood flow. There is some evidence indicating that patients with cerebrovascular disease may have impaired autoregulation, a condi-

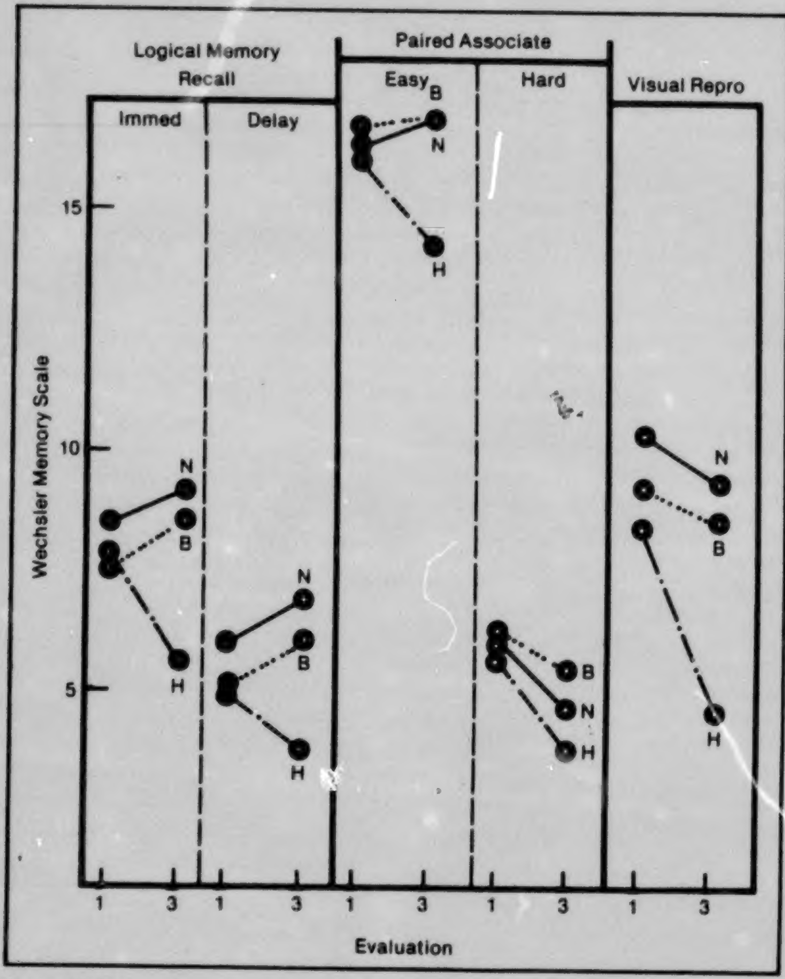

Figure II-17.

Wechsler memory scale mean scores on tests 1 and 3 among individuals initially examined at ages 60 to 69 with either normal (N), borderline elevated (B), or heightened (H) diastolic blood pressure on the initial examination.

tion which may last for days or weeks following an ischemic episode. Vascular insufficiency can develop when tissue perfusion pressure drops in relation to vascular resistance. The resulting reduction in blood flow may then give rise to ischemic hypoxia.

Degenerative changes in the brain may occur following subacute, prolonged, or intermittent ischemia (accompanied by hypoxia) which may lead to altered cognitive functioning. There is some evidence to suggest that reduced brain metabolic needs may be accompanied by lowered cerebral blood flow which is adjusted to meet the lowered cerebral energy needs (*52*).

There is evidence indicating that even minimum asymptomatic arteriosclerosis is associated with reductions in cerebral blood flow among elderly men who were living and functioning in the community (*52*). Among these subjects, the cerebral circulation and metabolic functions were almost identical to those observed in elderly patients with chronic brain syndrome and psychosis, except that the cerebral oxygen consumption in the group with minimal arteriosclerosis was still maintained at normal levels. The reductions in cerebral blood flow were associated with impairment in performance on a variety of tasks among those with minimum arteriosclerosis. The investigators suggested that the presence of vascular disease appeared to be associated with more rapid deterioration of mental functions, probably due to cerebral vascular insufficiency.

As noted by Obrist (*45*), there is a need to know more about cerebral hemodynamic and metabolic variables as they relate to cognitive functioning. With continued development and refinement of techniques, investigations on the pathophysiology of circulatory disorders may provide valuable information into the aging processes in the brain. Another area in need of further investigation involves the autonomic influence on cerebral blood flow. As discussed by Scheinberg (*53*), it is difficult at this time to draw any conclusions but recent evidence tends to suggest that cerebral blood flow may, in part, be influenced by alpha-adrenergic activity. Thus, there is some evidence that cerebral vasoconstriction may be mediated by norepinephrine, either exogenous or endogenous, and that the vascular receptor is alpha-adrenergic as indicated by drugs that compete for these sites and effectively block the action of vasopressors.

It has also been suggested that sympathetic nerves may have an influence in the mechanism of CO_2 action on cerebral vessels and have a role in cerebral spinal fluid production. Hypothalamic blood flow in the rabbit was reportedly increased by the administration of catecholamines and blocked by the antihypertensive agent, propranolol. Although as pointed out by Scheinberg (*53*), evidence is beginning to suggest that sympathetic innervation may have some influence on cerebral blood flow, the role of sympathetic innervation of cerebral vessels has yet to be resolved.

Psychosocial stress.—The effects of situational stress upon performance among hypertensives has not been systematically examined (*9*). Most investigators interested in the relationship between BP and cognitive functioning, however, have questioned the role, if any, of anxiety upon their findings of performance decrements among hypertensives.

The interest in the influence of stress in hypertension and performance stems, in part, from distinct bodies of literature. It is known that there is a relationship between sympathetic nervous system activity, the adrenal gland, and BP regulation. More recent findings (*23*) indicate that specific catecholamine containing nuclei in the brain stem and hypothalamus are also involved in the regulation of BP. Numerous investigators (*54–59*) have observed that hypertensives show sympathetic hyperreactivity to both physical and psychological stimuli. Many of the antihypertensive drugs lower BP by influencing the disposition of catecholamines in various ways or by blocking the receptors upon which they act (*23*). Interestingly, Shapiro and colleagues in a series of studies (*60–62*) found that many of the antihypertensive medications were effective in lowering BP to normotensive limits under basal conditions but BP frequently returned to hypertensive levels when individuals were exposed to both physical and psychological stressors. Thus, if hypertensives should experience greater situational anxiety in the psychological testing situation than do normotensives, then their performance may be adversely affected even when they are on hypotensive medications.

As noted in a review of the relation of environmental factors to systematic arterial hypertension, Benson and Gutmann indicate that ". . . the concept of a 'hypertensive personality' remains ambiguous and lacks both experimental support and theoretical meaning" (*63*). Much of the early literature of personality differences between hypertensives and normotensives was based upon subjective reports, making it difficult to objectively replicate the findings. In addition, since the personality measures were obtained after the high blood pressure was clinically observed, it is difficult to determine whether the behavioral characteristics were present before the disease process began or whether they were a consequence of the disease.

The findings (*9*) on coronary heart disease and behavioral characteristics may be relevant to the study of hypertension since high BP is a major determinant of heart disease. Major contributions in this area have been made by Rosenman and Friedman (*64*) in a prospective study of factors leading to the development of coronary heart disease. In a series of reports, they have described a specific overt behavior pattern (type A) associated with a high prevalence of clinical coronary heart disease. The type A behavior pattern has been characterized by excessive drive, aggressiveness, ambition, involvement in competitive activities, an enhanced sense of time urgency, frequent vocational deadlines, pressure for vocational productivity, restless motor mannerisms and staccato style of verbal response. The converse of the type A pattern was called type B. Individuals free of coronary heart disease but possessing the type A behavior pattern show biochemical changes similar to those observed in individuals with manifest heart disease, including elevations in serum cholestrol, fasting triglycerides, and serum pre-beta lipoproteins, accelerated blood coagulation and higher daytime excretion of catecholamines in the working milieu (*9*).

Abrahams and Birren (*65*) observed that men aged 25 to 59 years of age who appeared to be free of cardiovascular disease but possessed the Rosenman and Friedman (*64*) type A coronary-prone behavior pattern had significantly longer response latencies in both simple and choice reaction times and were disproportionately slower in choice response speeds than type B subjects. Birren (*66*) suggested that while age appears to be accompanied by a slowing in response speed, it is exacerbated by the presence of disease, particularly those diseases of a stress character which involve the sympathetic nervous system.

Krantz, Glass, and Snyder (*67*) examined the relationship between stress and performance in young individuals who had been classified as possessing either the type A or B behavior patterns. In their study, half of the subjects were subjected to the stress of loud noise bursts from which they could escape. The other half could terminate each burst by manipulating appropriate switches. After the pretreatment series, the same noise was again delivered to the subjects, all of whom could escape or avoid the noise by making an appropriate response on a shuttle box. The type A's performance was superior to the type B's under controllable, moderate stress but they performed less well under high stress conditions. The investigators suggested that the type A's appeared threatened by perceived lack of control over more intense environmental stressors and therefore gave up efforts to attain control. Further the type A's rated themselves as reliably more helpless and presumably more threatened by lack of control in the high stress-no-escape than in high stress-escape conditions. It was suggested that there may be a link between reactions to uncontrollable stress and a coronary-prone behavior pattern. At this time, one can only speculate as to the conditions under which hypertensives experience stress but it appears that they react physiologically to both physical and psychological stressors to a greater extent than do normotensives. The effects of situational stress upon the hypertensive's performance have yet to be systematically examined to determine whether it may, in part, account for the performance decrements associated with hypertension.

Future Research

Throughout this paper, an attempt has been made to point to areas where future research is needed. Other areas are discussed below.

1. Since alterations in cerebral blood flow and metabolic needs may play a major role in the performance decrements observed among hypertensives, it is important to examine these factors in the *same* subjects. The effects of antihypertensive drugs upon performance and cerebral circulation should be examined for acute as well as chronic effects. There is a need to know whether there are differential drug effects such that some drugs may be better in controlling BP while at the same time having minimal effect upon cognitive functioning.

2. The side effects of drugs, such as sedation, depression, etc. should be examined in relation to cognitive functioning since these factors alone may impair performance. As noted by Wang and Busse (*46*), the effects of other widely used drugs, such as tranquilizers and antidepressants, that tend to lower BP should be examined to determine their effect upon performance.

3. Alternatives to drug usage in lowering BP, such as biofeedback training and meditation, should be studied to determine whether re-

ductions in BP can be maintained at normotensive levels for long periods of time and whether improvements in performance will be found as the findings reported by Goldman and colleagues (*42*, *43*) suggest.

4. Other risk factors associated with hypertension and heart disease, such as diabetes, smoking, diet, cholesterol levels, etc., should be considered since these variables may possibly affect performance independent of cardiovascular disease. As noted by Spieth (*7*), although the findings are contradictory, there is some indication that reductions in cholesterol levels are associated with improvements in performance.

5. Prospective studies employing longitudinal as opposed to cross-sectional research strategies may be particularly useful in detecting differential rates of decline in performance among hypertensives which, in turn, could be linked with pathological changes over the same period. Since there is an increase in the incidence of multiple chronic diseases with increasing age, life span studies should be encouraged. In that regard, there is some evidence that children with high blood pressure may be at greater risk of developing hypertension (*68*) and it would be interesting to determine whether BP is related to performance among the young.

6. Since there is a growing interest in sex differences in cognitive functioning (*69*, *70*), it is important to note the sex differences in incidence of hypertension and coronary heart disease particularly among the middle-aged. One could speculate that differences in health status may, in part, account for the performance differences found between men and women (*71*).

7. Epidemiological studies indicate there are sex, racial, as well as socioeconomic differences in the incidence of hypertension and it is important to take these factors into account in behavioral studies since these factors alone (*63*) may account for the differences in performance observed between hypertensive and normotensive individuals.

8. Since the dangers of sustained hypertension are generally described in medical terms (e.g., increased risk of heart disease, strokes, and kidney impairment) it would be interesting to determine whether including intellectual impairment as an additional risk factor (even during the middle-aged years) would serve as an incentive to hypertensives to stay on their prescribed drug management programs. There is also a need to alert patients to potential side effects of certain antihypertensive medications that may affect mental acuity, since the modification of their drug routine may eliminate such problems.

9. Although it has been widely speculated that stress associated with the psychological testing situation may have a greater adverse effect upon the performance of hypertensives than on normotensives (*7*, *9*), this is an area that has not been carefully examined. In such studies, self-reports of anxiety, life stress questionnaires, as well as arousal as measured by psychophysiological techniques should be included.

10. In view of the complexity of the hypertensive disorder, there is a greater need for multidisciplinary research such that behavioral

measures can be evaluated in terms of etiology and severity of the disease, as well as to take into account possible differences in neuroendocrine activity which may be valuable in interpreting the variability in performance often found among hypertensives.

11. Since there are frequently age-related changes in responsivity to drugs, the effects of antihypertensive drugs in the elderly should be examined carefully both in terms of physiological and cognitive functioning, with drug blood levels taken into consideration. The age effect should be considered in studies involving antihypertensive drugs, since the problems and questions that need to be considered may be quite different among the elderly than in middle-aged individuals.

12. Increased attention should be paid to the physical health status of subjects in the studies of cognitive functioning among middle-aged and elderly individuals. During the past 12 years, less than 20 percent of the behavioral studies in aging even mentioned the health status of their subjects with this percentage declining during the past 6 years (*69*). The findings reported here, however, suggest that the pattern of differential rates of decline in cognitive functioning may be related to health status. An important issue is whether antihypertensive drugs, which reduce the risk of dangerous cardiovascular consequences, will also reduce the risk of cognitive impairment.

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Discussion

Ostfeld: The data from the National Center for Health Statistics show that high blood pressure can occur in any segment of the American population, but that it has a much higher prevalence among people of rural origin and of people with limited educational and occupational attainments than in any other groups. Can you relate that in any way to the findings in the literature that you reviewed? Do you think that may explain, or at least may potentially explain, some of the relationships observed in the literature you reviewed in the earlier part of your presentation?

Wilkie: You have raised a very important issue and one that has received very little attention in the literature focusing upon cardiovascular pathology and cognition. It is highly likely that many of the cultural and psychosocial variables which are associated with the prevalence of high blood pressure are, in part, accounting for a portion of the performance decrements observed among hypertensives as a group. For example, in the Duke longitudinal study noted earlier, the hypertensives who did not return for subsequent evaluations performed less well than the remaining groups and attrition in that study was related to education and socioeconomic status. On the other hand, the hypertensives in the Duke study who completed the longitudinal study were not statistically different from their counterparts with respect to race and socioeconomic status and initial psychological test scores, but over the 10-year followup period they did show the greatest decline in performance. Thus, it is extremely important in studies of cardiovascular disease and cognitive functioning to consider the subject characteristics of the sample with respect to genetic, cultural, and psychosocial factors.

Ostfeld: I did not mean the data in the Duke Aging Study; I mean the literature in general.

Wilkie: I thought the Duke study was a good place to start. In 1966, those investigators found that reaction time among older individuals with and without the presence of cardiovascular disease was related more to socioeconomic status than to cardiovsacular pathology. I recall that Hertzog and his co-workers found that when the effects of socioeconomic status and income were statistically controlled, some specific types of intellectual abilities were no longer related to cardiovascular disease, whereas other types of intellectual abilities continued to be linked with cardiovascular pathology. Undoubtedly, the relationship between hypertension and cardiovascular diseases and cognition is a complex one and there are many variables that should be controlled. Two other important factors that should be considered are stress related to the testing condition and drug effects upon performance. For example, in Spieth's study, you will recall his untreated hypertensives were undergoing medical certification by the Federal Aviation Administration while the treated hypertensives were not. Thus, as Spieth noted, it was possible that the untreated hypertensives may have experienced greater stress during the psychological testing than did the treated hypertensives, making it difficult to interpret the effects of antihypertensive drugs upon performance among his hypertensive subjects. Further, few studies have controlled for the effects of antihypertensive drugs upon performance.

Kannel: It seems to me to be critical in this to dissociate the effects of blood pressure from the effects of treatment because many of the drugs which are used to treat hypertension have cerebral effects. Many patients taking antihypertensive agents complain that they become lethargic and it is hard for them to keep up with their professional activities. In some cases, as you noted, people become depressed which in itself can adversely affect performance.

I would like to see what effects these drugs have on normotensive people—what it does to their performance. This would provide baseline data for these other studies. It might also be useful to look at people maintained on different kinds of antihypertensive agents at a given level of lowered pressure.

Cohen: Do you have information on the drug status of the people you were studying?

Wilkie: No. In the Duke longitudinal study the evaluations were initially about every 2 to 3 years and it was not possible to control drug usage. However, if the hypertensives who completed the 10-year followup study were on antihypertensive medication, their blood pressure was not adequately controlled since most of them continued to have elevated blood pressures on subsequent evaluations. There are very little data available on the acute or chronic effects of antihypertensive drugs upon cognitive functioning among hypertensives, with most of the information on drug effects stemming from clinical reports of side effects of antihypertensive drugs. Thus, relatively little is known about the effects of the antihypertensive drugs upon cognition among the majority of hypertensives. There is a need to examine the effects of antihypertensive drugs upon performance according to the drugs' mechanism of action.

Weksler: I think your point is very well taken because I seem to remember one study where the impairment in cognitive function was related to older people being poor test takers and the parameter that I think was measured was pulse rate. The pulse rate went up during these tests in older people to a much greater extent than in younger people.

Then, they went on and gave those people propranolol. These were normotensive older people. They found that with the drug, test scores seemed to improve. This was an important control population because it was nonhypertensive. I suggest that some of the drugs that are used would influence the results so that a control population is critically important.

I was wondering whether the hyperresponsiveness of both a hypertensive population and an older population may be the common thread in both. Their falling cognitive function may be related, in part at least, not only to the change in the caliber of the vessels to which you referred, but also that hyperactive sympathetic responders do not take tests very well. Old people may come from that population and hypertensives may also come from that population.

Wilkie: Most of the work you have referred to was completed by Dr. Carl Eisdorfer and myself. During the past 10 years or so we have been examining verbal learning performance in relationship to stress or physiologic arousal as measured by indices of autonomic nervous system activity among healthy old and young individuals. Our findings indicate that heightened arousal among old men and women is associated with poor verbal learning. In one study (*36*) we found that very healthy old men who were given 10 mg propranolol intravenously showed a decrease in heart rate and free fatty acids accompanied by learning performance which was superior to that of a control group given a placebo. At this time, this line of research has not been carried out with older hypertensives.

Weksler: Well again, I do not recall whether it was in your paper or your work, but it went on to suggest that if you take away time pressure "cognitive impairment is lowest."

Wilkie: In general, old people show a slowing in reaction time and they tend to perform less well on tasks involving a perceptual motor component and on time cognitive tasks where they must perform very rapidly. This pattern of decline appears to be exacerbated among individuals with cardiovascular disease and occurs even among middle-aged individuals. Whether it is related more to stress or alterations in cerebral blood flow, or a combination of both is not known at this time.

Speaker: Do you have any ratings of these individuals, the normotensives and the hypertensives, outside of the experimental situation so you can evaluate their normal functioning in the environment. Is being on drugs affecting them in a normal situation?

Wilkie: In the Duke longitudinal study, there was an extensive battery of tests including the psychological measures of performance as well as psychiatric evaluations, psychosocial measures, physical examinations and clinical laboratory studies. We did not, however, examine the relationship between hypertension and social or psychiatric adjustment. As I indicated earlier there are little data available on the effects of the antihypertensive drugs upon behavior among hypertensives.

Gruenberg: When you reported these drops in cognitive performance in hypertensives, you were talking about average scores of hypertensives dropping as compared to average normotensives not dropping. How about the distribution? What proportion of the hypertensives showed a drop?

Wilkie: Among our subjects, the variability in performance among the hypertensives was slightly (but not significantly so) greater than that observed among their age peers with lower blood pressures. Task complexity and the nature of the abilities measured were important factors, with some, but not all, hypertensives showing a decline on the very simple tasks while on the more complex tasks most hypertensives would perform poorly. Some decline over the 10-year followup was also observed among some individuals with normotensive and borderline elevations of blood pressure, particularly so among those who were 70 years old and over at the initial testing.

Gruenberg: You did see some decline in the other groups?

Wilkie: Yes. Cognitive impairment in old age appears to be related not only to vascular alterations but also to primary neuronal changes. I think an important issue at this time is whether the incidence of cognitive impairment associated with vascular changes can be reduced if hypertension were adequately treated.

Epidemiology of Injury in Older Age

Carol C. Hogue, R. N., Ph. D.

Department of Nursing
Duke University Medical Center
Durham, NC 27710

Injury and disease alike have been considered by primitive peoples to be the visitation of outraged spirits or the effect of human misbehavior directed through some evil eye. More than two thousand years ago Hippocrates provided alternatives to the power of ghosts, witches and demons in the cause of disease through his systematic account of the effect of environment on health. To a certain extent Hippocratic thought influenced ideas about disease until the advent of the germ theory dominated thinking about disease; only in the last two decades have we considered multicausal approaches to disease and multivariate approaches to its control. Epidemics of infectious disease prior to this century probably allowed for little consideration of injury, and certainly the germ theory, with its emphasis on an infectious, external agent left injury as "accidental happenings" without any rational, observable explanation. Injury or so-called accidents came to be viewed as a health problem in the 1930's and 1940's; it has never in any sustained way been studied with the same rigor accorded disease.

The word "accident" will continue to be used commonly by lay people, but epidemiologists have, by and large, stopped using that term, replacing it with injury or injury events (*1*). Definitions of accidents refer to random or chance events, yet existing data indicate that accidents, like diseases, are nonrandom events. Epidemiologists are concerned with the identification and understanding of nonrandom patterns of events. The term accident also has the connotation of bad luck, carelessness, sinfulness, acts of God, and other moralistic terms which tend to exclude scientific approaches to this problem.

Much of the work in this field has been largely atheoretical, but some interesting conceptualizations have been offered in recent years. Before we turn to injury in older people, I'd like to mention some of this conceptualization.

Although John Gordon's paper in 1949 (*2*) encouraged a rash of so-called accidents studies with a long list of causative "agents" such as cars, guns, knives, medicines, stoves, rugs, stairs, etc., the work of King (*3*), Gibson (*4*), Haddon (*5*), and Waller (*6*) suggest that the agent in all injuries is physical energy in one form or another (kinetic, chemical, thermal, radiation, electric), that most previously identified agents were only vectors, and that "excessive levels of energy exchange or, rarely (as in drowning or poisoning) excessive interference with normal patterns of energy transfer produce tissue damage" (*7*). Three phases in the occurrence of injury and its outcome are identified, "the pre-injury phase, which includes the events that lead to the unintentional dislocation of energy from its usual setting, the injury phase in which energy is transferred to human tissue, and the post-injury phase in which the adequacy of emergency and follow-up care often play an important role in determining ultimate outcome. . . A brief review of the pre-injury phase will help focus on the nature of the problem. . . . Basically, the process of dealing with energy sources in our environment depends on two factors, the performance level of the person and the task demands involved in using the energy (*1*).” The task of walking from one room to another is less demanding than is the task of walking across a busy city street. In either type of task, however, it is only when the demands of the task exceed the performance level that a harmful exchange of energy occurs.

Once energy is released the occurrence of an injury is dependent upon the amount of energy released, its rate of transfer, its distribution over body tissues and the nature of the body tissue itself. Thus “tissues capable of spreading the load, such as the pliable bones of young children resist serious injury more effectively than do tissues that localize energy, such as the brittle bones of the elderly.” (7).

In the post-injury phase both the nature of the injury and the adequacy of emergency and definitive care determine survival and amount of promptness of recovery. A graphic representation of Waller's model appears in figure II–18.

Epidemiology is concerned not only with the understanding of determinants of abnormal conditions or health states, but also with identifying and testing methods for control of abnormal conditions and maintenance of health. Haddon (*8*) and Baker (*9*) have suggested categories of countermeasures as approaches to injury control based on the above model. Broad categories of countermeasures are: changing the environment, making tasks less demanding, strengthening people, and providing adequate emergency treatment.

Unintentional Injury

Unintentional injury is the leading cause of death in persons aged 1 to 38. Among persons of all ages, unintentional injuries are the fourth leading cause of death. In 1974 unintentional injuries were the fifth cause of death in persons 65 to 74 years of age, and the sixth leading cause of death for persons 75 and older. The death rate for unintentional injury per 100,000 persons (data for 1974 from National Center for Health Statistics) was 50 for all ages combined, 69 for 65 to 74 years, and 191 for persons 75 and older. Put another way, the older population is overrepresented in deaths due to unintentional injury: in 1975, 24 percent of all unintentional injury fatalities occurred to the 10 percent of the population who were 65 years and older.

It is not true, however, that a high injury death rate also means a high injury rate. In general, injury rates are lower in the elderly than in younger persons. In 1974, 32, 36, and 32 were the respective injury rates per 100 persons among children under 6, 6 through 16, and 17

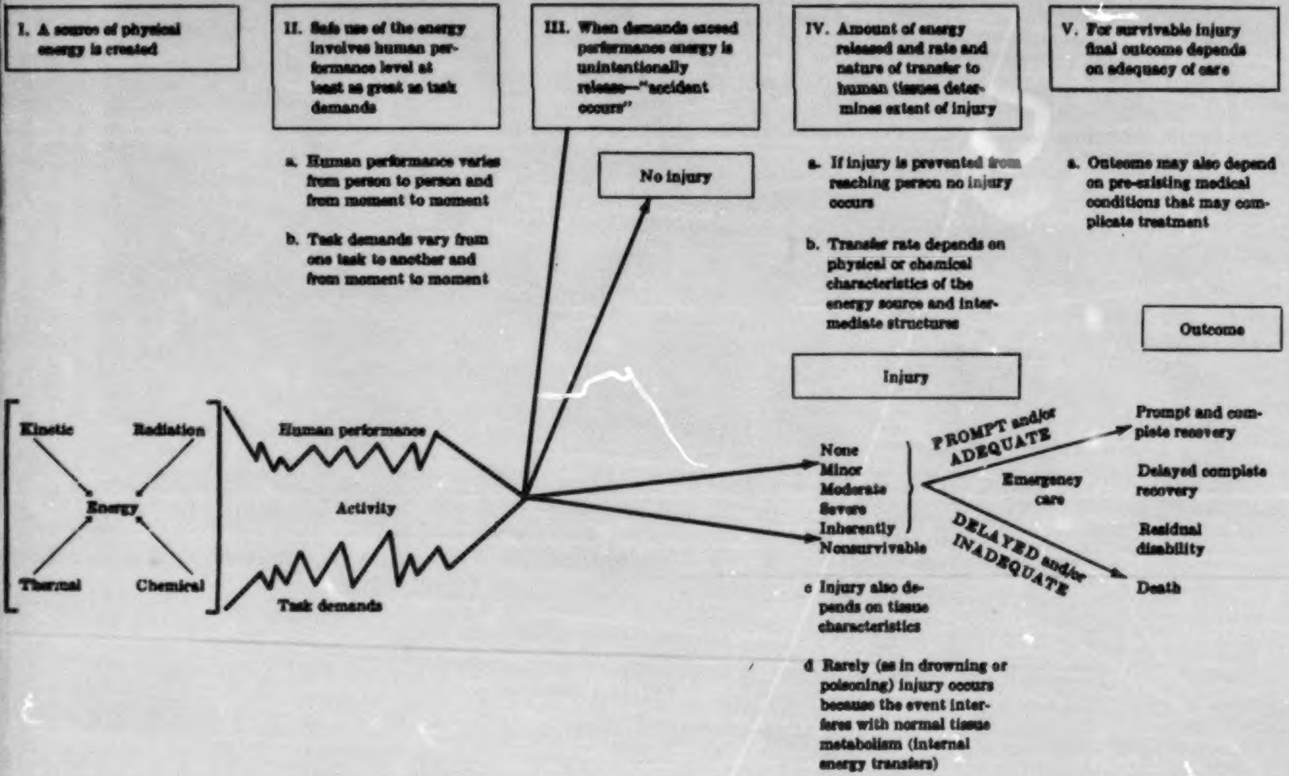
I. A source of physical energy is created
II. Safe use of the energy involves human performance level at least as great as task demands
III. When demands exceed performance energy is unintentionally released—"accident occurs"
IV. Amount of energy released and rate and nature of transfer to human tissues determines extent of injury
V. For survivable injury final outcome depends on adequacy of care
a. Human performance varies from person to person and from moment to moment
b. Task demands vary from one task to another and from moment to moment
No injury
a. If injury is prevented from reaching person no injury occurs
b. Transfer rate depends on physical or chemical characteristics of the energy source and intermediate structures
a. Outcome may also depend on pre-existing medical conditions that may complicate treatment
Outcome
Injury
Kinetic
Radiation
Energy
Thermal
Chemical
Human performance
Activity
Task demands
None
Minor
Moderate
Severe
Inherently Nonsurvivable
PROMPT and/or ADEQUATE
Emergency care
DELAYED and/or INADEQUATE
Prompt and complete recovery
Delayed complete recovery
Residual disability
Death
c Injury also depends on tissue characteristics
d Rarely (as in drowning or poisoning) injury occurs because the event interferes with normal tissue metabolism (internal energy transfers)

Figure II-18.

A model for the occurrence of unintentional injury and its outcome (Reference 6).

through 44, while the number of persons injured in the 45 to 64 age group was 19 and for 65 years and older, 17 (*11*).

Overall then, the elderly have relatively low injury rates compared to other age groups, but high case fatality rates. In addition, rates of days of restricted activity and of bed disability and hospitalization due to injury are many times greater in older people. Therefore, even though in absolute numbers injuries in older people are relatively few, the disability, dysfunction, utilization of health services and death rates given the event of injury are reason for further scientific efforts.

Three types of events account for 75 percent of all injury deaths among the elderly: falls, fires and contact with hot substances, and vehicular crashes, including those involving pedestrians. Let us consider each of those types of injury event, reviewing descriptive and analytical data available, and in each area, making suggestions for further research.

Falls.—Persons 65 or older account for more than 70 percent of the deaths due to falls, having both a higher than average number of falls and high case fatality rate. More than half the deaths due to unintentional injury in people 65 and older are due to falls (*12*). For all ages combined, the death rate for falls has gradually dropped from a high of nearly 20 per 100,000 in the midthirties to less than 7 in 1975 (*10*). Part of that decline is due to changes in methods of classification. The rate of deaths from all types of unintentional injury is highest for nonwhite males; after 75 white rates are higher than nonwhite and white females highest of all. Both injury and death rates for falls are higher in urban than rural areas. The states with the highest age-adjusted death rates for falls are in New England; Hawaii and Mississippi have the lowest.

The classic study on falls was published in 1960 by Sheldon (*13*). Sheldon described clearly the phenomenon of drop attacks, with no aura and no amnesia. He reported that 25 percent of the 500 falls he studied were drop attacks. Waller's studies (*6*) suggest that alcohol and medical impairment may lessen performance levels of persons who have falls.

A number of questions have been raised about osteoporosis and falls. Longitudinal studies of older women have shown substantially higher fracture rates among osteoporotics (*14*), and fracture incidence inversely proportional to bone mineral content (*15*). It has been suggested (but not demonstrated) that some frail elderly people have such brittle bones that a fall occurs *because* of a broken bone.

Further research in the epidemiology of falls should not only continue to help us understand more about why people fall but why they are injured when they fall. Research questions arising from the injury model presented earlier should be tested, and they should include the testing of effectiveness of countermeasures. Questions could address such diverse areas as prevention of falls in the first place with investigators paying attention to perceptual deficits in those older people who have the most falls, with research to evaluate the effectiveness of various environmental measures such as increased illumination with minimal glare. Most falls in older people occur in private dwelling units, public buildings, or residential institutions—all governed by building codes; consider the opportunity in the long run for environmental countermeasure if it could first be shown that specific environmental designs are associated with fewer falls. Questions arising from another category of countermeasure would deal with strengthening the individual to diminish the impact of falls. There is evidence that there are fewer fractures of the spine among people who live in areas with at least 3 ppm of flouride in their drinking water (*16*). There is the possibility that even higher levels of flouride may strengthen bones enough to decrease the incidence of fractures, but to my knowledge no definitive controlled study has been carried out. A number of physical fitness programs for older people have been started. It would be useful to compare over time the rates of falls and fractures in participants with the rates for their controls.

Perhaps more information is needed about the characteristics of surfaces struck and distance fallen. While it seems logical that less forgiving surfaces and greater distances fallen would increase the probability of injury, there is little convincing evidence to that effect.

An additional category of countermeasure, provision of adequate emergency treatment, suggests a need for additional research. How soon do injured people get emergency treatment? In a study of nonhighway injury emergency care (*17*), Waller has reported that people under the age of 30 preferentially receive assistance by lay-people and professionals.

Fires and contact with hot substances.—Seven out of 100 injury deaths in people 65 and older are related to fire or other hot substance contact. Among people over 65, death rates for males are much higher than for females, and relatively high for nonwhite persons of both sexes. The elderly are overrepresented in this category of deaths, with persons 65 and over having 29 percent of all fire and burn fatalities (*18*). Speculation about why the elderly are overrepresented includes the suggestion that they fall against hot surfaces, that they may be unable to extricate themselves from a fire because of arthritis or other functional disability, and once a burn injury occurs recovery may be complicated by the presence of chronic disease. In particular, the inhalation of smoke and the toxic fumes of burning synthetics are likely to be more difficult for persons with pulmonary or cardiovascular impairmant.

Waller (*18*) has studied the interaction of product and human factors in nonhighway injury fatalities. While there is little research in this area, statistical data suggest interventions to make the environment safer, such as building codes to make buildings less likely to burn and having flammability standards for fabrics used in upholstery and clothing. How much research effort ought to be directed toward either improving our understanding of the injury phenomena associated with burns or with countermeasures designed to decrease fires or their impact probably needs to be examined along with the research needs in the areas of falls and vehicular crashes.

Vehicular crashes.—Compared to what we know about other kinds of unintentional injury in older people we know quite a bit about road injuries. This knowledge has not contributed much yet toward improving the quality of life for older people.

There are 11.4 million licensed drivers 65 and older. They represent nearly 9 percent of all the licensed drivers in the United States (*10*). The proportion of drivers 65 and older is rising in spite of tightening of reexamination requirements in many states. The proportion of female drivers decreases sharply with age when cross sectional data are reviewed; as more women begin driving early that pattern is likely to change. Older drivers drive less. Planek reports that "drivers in their fifties begin to show a decrease in annual miles driven that continues steadily in later years" (*19*). Opposing conclusions have been drawn about the safety of older drivers. It is true that older drivers have a relatively small number of crashes; it is also true, however, that older drivers have a higher crash rate than any group except the under 25 age group if miles driven or exposure is taken into account. Since drivers and not liabilities are licensed, the small absolute number of crashes by older drivers bears remembering.

In 1975, 5,700 people 65 and older died of vehicular crashes. That represents 12.3 percent of such fatalities for all ages, and nearly 23 percent of all injury fatalities for people 65 and over. Two thousand, or more than one-third of those vehicular crash fatalities, were pedestrian deaths.

What do we know about the characteristics of vehicular crashes of people over 65? In a study of 354 male and female white drivers aged 46 to 71, who were panelists in the Duke adaptation study (described elsewhere) (*20*), the descriptive findings are similar to those reported for older drivers in other studies. Motor Vehicle Department records for a 3-year period were examined; Adaptation study data on physical health, psychological and social characteristics were also studied. For the 3-year period, slightly more than 15 percent had one or two crashes. That incidence yields for 1 year an average rate of 0.056, which compares favorably with the North Carolina rate of 0.063 for drivers 45 and over for a concurrent year (*21*). Only 16 percent of the drivers in this study (28 percent of the men and 3 percent of the women) estimated their annual mileage at 15,000 or more. There was no association between crashes and estimated miles driven, but more than one-third of the crash drivers said they drove less than 6,000 miles per year. None of the crashes occurred between 9 p.m. and 7 a.m., and 81 percent occurred between 7 a.m. and 6 p.m. Only 12 percent of the crashes took place in rain, and none in snow. Eighty-two percent of the 56 crashes took place in the driver's home county and an additional 7 percent in the adjacent county. Most (79 percent) occurred in business or residential sections. Twenty-nine persons were injured in 14 crashes; there were no immediate or delayed fatalities. All but two crashes involved two or more vehicles. The predominant error recorded for these crashes was disregarding a stop sign or traffic signal or failing to yield (25 percent). Only one driver was charged with excessive speed associated with a crash. In only 2 of 56 crashes was the driver noted to be under the influence of alcohol with ability impaired. In this study no association was found between vision, hearing, cardiovascular or musculoskeletal impairment, general functional status, life change or coping resources and road crashes (*22*). Other studies have also addressed questions related to sensory deficits and medical impairments in older drivers.

Planek (*19*) reviews literature which documents narrowing of the visual field beginning in the late thirties, the need for increased illumination rising with age, glare sensitivity increasing beginning at about age 40, and the rate of dark adaptation decreasing with age. Studies which have attempted to bridge the gap between those laboratory findings and actual driving performance have been very few in number and have had conflicting results (*23*, *24*). Planek cites research showing deficits in short-term memory, visual discrimination, and logical interpretation of stimuli (*19*), speculating that those deficiencies probably affect driving.

Waller (*25*) found that drivers known to have diabetes, epilepsy, cardiovascular disease, alcoholism and mental illness averaged twice as many crashes per 1 million miles of driving as control drivers with no known disease (data age-adjusted). Crancer and McMurray (*26*), on the other hand, found no excess of crashes among drivers with a heart disease license restriction; they did find higher crash rates for drivers with epilepsy, fainting and other medical conditions at all ages. Planek cites work by Grattan and Jeffcoats (*19*), using British and Swedish data, showing incidence ratios of only 1 in 1,000 crashes associated with sudden illness. In the Baker and Spitz study of fatally injured drivers (*27*), ateriosclerotic heart disease by autopsy was found with similar frequency in those drivers who were found at fault and those who were not.

Since persons over 65 (as well as preschool age children and alcoholics in the middle years) are overrepresented in pedestrian fatalities, some attention to pedestrian injuries seems indicated. The death rate is much higher for males than females and slightly higher for nonwhites than whites. Iskrant shows data to support the statement that the pedestrian death rate is lowest for married persons and highest among widows and widowers (*12*). In a classic study of pedestrian injuries by Haddon and others (*28*), those who were injured and killed were, on the average, 10 years older than those injured who survived.

Shifting back to drivers, we note a similar finding by Baker and Spitz (*27*). In a study of 328 fatally injured drivers, using autopsy records and police records, they found that the proportion of drivers 60 years or older was five times as high among those killed as among drivers who survived crashes, and further that "delayed death" was more common among older drivers and was associated with less serious injuries than in younger drivers.

Where does this information about pedestrian and nonpedestrian motor vehicle crashes lead us with respect to further research? Perhaps additional documentation of the relationship of perceptual decrements and medical conditions is in order. Beyond that, however, our further research should have more payoff than has the careful documentation of high risk groups in the past. Returning once more to categories of countermeasure which can guide further research, we will make a few additional suggestions. An example of making tasks less demanding would be research leading up to reducing the complexity of the driving task; that would first require studies in test driving or simulator conditions to describe the driving task for older persons of varying characteristics. To my knowledge older drivers have not been used as test drivers either to influence product design or to define the driving task. With respect to environmental change, studies of transportation needs of the elderly will be required before informed policy decisions can be made about licensing older drivers. Evaluation of perceptual aids to older drivers might be indicated. The consequences of crashes and other types of injury to older people needs to be studied. It has been suggested that an injury event for many older people is the abrupt transition from independence to dependence for life.

Summary

In summary, then, injury has been discussed as a nonrandom event of considerable consequence to older people who have high case fatality rates and high dysfunction and disability rates from falls, fires and contact with hot substances, and vehicular crashes to pedestrians and drivers. A model for the injury event was presented, including categories of countermeasures. On the assumption that identifying and testing of measures for control of health problems is the legitimate concern of epidemiology, and with the knowledge that many times in the past understanding of environmental and personal influences have led to effective preventive practices even before cause could be clearly assigned, this presentation has provided only limited support for continuing identification of high risk groups and much greater support in the direction of more applied research directed toward evaluation of countermeasures to reduce injury losses in older people.

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Discussion

Siegel: Among the aged, the difference in mortality from motor vehicle accidents between the sexes is almost the highest for the leading causes of death. I think it is a terribly important area for research to find out why the men get killed much more often than the women, even if you adjust in some way for hours of driving exposure.

Hogue: I would like to look into that. I believe that if there is some sort of control for mileage, the difference washes out.

Siegel: Miles as passengers or miles as drivers?

Hogue: It is very hard to get those estimates, but I think that sometimes the very best we can get is miles driven as the driver of the vehicle, but we all know that passengers and pedestrians are also at risk.

Siegel: It is important. In the effort to divide or partition the genetic and environmental factors, we tend to think of the accident area as one that is essentially environmental even though we should talk also about the difference in the physiological ability to stand blows, in reaction time, in the acuity of the senses, et cetera.

Hogue: I think we really have to find ways of looking at both, the environmental effects and the individual capabilities.

Costa: Dr. Hogue, your emphasis on the task of redesign seems to stress man-machine design facets. I wonder if you would evaluate the role or contribution of the driving style of the elderly; for example, cautiousness versus the sensory motor difficulties

Hogue: I am saying that we do not know much about the task as it is perceived by older people For example, whenever there is test driving done for the sake of product design, when General Motors has test drivers come to help them figure out where to put instruments on the instrument panel and that practical kind of thing, they usually have young males. They never have old people of either sex and I think that seeking a redefinition of the driving task seems to get back to the basic levels of defining what the driving task is for older people. In response to your question about some of the characteristics of drivers—these are noted when drivers are cited with violations, which by and large are errors of omission, failure to yield, difficulty getting from one place to another, and not problems of high speed that other drivers have. If your question suggests that which sometimes has been raised, "Do older people cause accidents because of the way they drive?" I think that is perhaps not a researchable question. If the question implies there is something someone can do to cause an accident without actually being a part of it, I do not know any way to study that.

Costa: Do you really think a design engineering task is the problem or do you think that there is any profit in looking at driving style?

Hogue: I think that the driving styles probably would make a difference. We should stop and think about what might influence that. If your vision is 20/120, you are going to have a very different driving style when you are getting ready to do something like read a sign to know which lane to get into, than if you have 20/20 vision. The design of road signs is set for people with 20/30 and 20/40 vision; 20/30 by one group of engineers and 20/40 by the other. I think that does make a difference and it brings into play the whole area about which we need to know more. In applied settings, I think it is useful to know that dark adaptation decreases with age, but it is more useful to know what relevance that has to people who drive.

Siegel: Very often, when we are driving on a highway, we find that the person who is pulling out into the highway from a driveway at a slow speed is an elderly person. Now this may be related to driving style and for the inability of the driver to judge the speed of the passing vehicles or even his/her own speed.

Sacher: Is there a cohort factor in driving skills?

Hogue: Certainly one of the cohort factors in the general population sense is when people begin driving. We know that the cohort that is dying off now is probably the last cohort, or close to the last large cohort of people, who did not drive fairly early after they could drive at the age of 16 or 17. We also know that if people begin driving later, they have more difficulties for various reasons in being able to pass drivers' license examinations for the first time.

Speaker: In all these accidents or injuries, you stress that they are not productive and that we should concentrate on the engineering features and promote them. I would suggest that another thing you could stress is that old people are educable and we should try to work on the person as well as the environment.

Hogue: I do not think that I have made myself as clear as I had hoped to. I really did not mean to stress the engineering aspect. I really meant to encourage looking at both environmental demands and individual personal capabilities. I think we have been through an era that has been identified by some people who do work in this field all of the time. I do not blame the victim, in which the strategy has been directed toward education and having scare messages and defensive driving courses. There is a strong discontent with continuing along these lines. It does not mean not to try to improve the perceptual capacity of drivers or to do some education, but not to be content with that, and to think about what environmental things we can do that do not rely on the participation of individuals. That is what I meant to say, and I am glad I have had this chance to say it.

Costa: I would like just briefly to take issue with the question of the nonrandom nature of injury events. There certainly is a kind of contingent nature to these events with some meaning to the word accident.

It is certainly not to be inferred that all we need to do is know enough determinative factors and they all will be entirely predictable.

Hogue: I do not disagree with you; I think that was precisely one of the points that I was making. The mere identification of high risk groups or subgroups does not do anything to help us prevent injuries from happening. However, it would be profitless to even study in this area if it were not true that injury is nonrandom. If it is a random event, why bother, it is going to strike us like lightning or bad luck.

Costa: Causal factors can depend on a great many extended factors.

Hogue: I think our ability to add to control is dependent upon a certain amount of predictability or nonrandomness.

Closing Remarks by Chairperson George A. Sacher

As chairperson, I am in the interesting situation of being a laboratory biologist dealing with nice neat experimental problems who is thrust into the areas of human epidemiology. It has been very mind-expanding to learn what some of the biomedical correlates of human aging are.

The problem of relating the many diverse sociopsychological manifestations of aging to the biomedical substrate of the organism, which we are talking about today, is probably the most important problem that biomedical and social gerontologists can consider. In that regard, this kind of interdisciplinary experience is very important for all of us.

Session III

Social, Psychological and Functional Correlates of Aging

Opening Remarks by Chairperson Judith Cohen, Ph. D.

Throughout the day, I have been intrigued that sessions reporting research on biological correlates of aging have also been emphasizing the necessary complexities of the aging process, in which biological, psychological, and sociocultural areas are mixed. Whether we are concerned with longevity, disability, or specific causes of mortality like CHD or suicide, our understanding of these outcomes depends not only on biological information, but also, as we heard earlier, on information about the process of survival in an environmental context. However, I see no problem with continuity of some of the ideas we have already discussed and the ideas we will be discussing in this session. The continuity might best be seen in terms of those environmental contingencies upon which continued survival depends. Some of those contingencies, as we have heard, are in the physical environment; others are part of our social and psychological environments.

A continuing problem, however, is that asserting that all of these disciplinary areas are relevant in our understanding of aging does not help us to see how these areas of understanding can be articulated. Perhaps increased involvement of epidemiologists in the study of aging can help here. Our working definition of epidemiology bears out the fact that epidemiologists have always had to work with diverse types of people, to help them communicate with each other, and to conduct interdisciplinary research of one kind or another. It is just possible that some progress can be made in that direction with this meeting too.

Aging and Suicide: Reflection of the Quality of Life?

Robert C. Atchley, Ph. D.

Scripps Foundation Gerontology Center
Miami University
Oxford, OH 45056

Suicide is an offensive but fascinating subject to Westerners who see preserving our minds and bodies as a central goal of life. We have a deep-seated drive to learn *why* suicides occur; why some of us quite purposefully reject what the bulk of us value most, life itself. As a topic, suicide holds a fascination for both professionals and the general public far greater than one would expect from a cause of death that seldom accounts for more than 1 to 2 percent of the total deaths in a population. A recent bibliography (*1*) lists nearly 5,000 references on suicide, spanning 1897 to 1970, most of them clinical studies concerned with why people commit suicide. Through techniques such as the psychological autopsy it is possible to accumulate information on the suicide apart from what is given on the death certificate (*2*).

There have been two basic approaches to the study of suicide. The first involves clinical studies of suicides; the second involves the use of official demographic statistics. Neither approach is entirely satisfactory. Clinical studies usually have no way to place suicide into a context that involves nonsuicides as well as suicides. Demographic studies, on the other hand, suffer from a lack of in-depth information on both suicides and nonsuicides. Because suicide by definition requires knowledge of the intent of someone who is dead, both types of studies suffer from inadequate operational definitions of suicide. Both clinical and demographic studies are required, but the sum of both types is still far short of the ideal in terms of our knowing who commits suicide and why.

There is a good bit in the clinical literature about the relationship between age and suicide. However, most of this literature simply assumes that suicide rates are high among older people and proceeds from there to speculate about why age is related to suicide. Suicide among the elderly has been tied to bereavement (*3*), serious physical illness (*4*), hopelessness (*5*), depression (*6*), retirement (*7*), and organic brain disease (*8*). Some of these explanations turn out to be either spurious or too vague to allow testing. For example, it is true that suicide rates for males are higher following age 65 than they are earlier. But this cannot be used to establish a link between suicide and retirement because suicide rates for males increase *in a straight line* from age 15 to 85. There is no sudden surge at age 65 or at any other age for that matter.

The demographic literature on suicide contains a few cross-national studies of suicide rates (*8*, *9*, *10*). Only Kramer et al. (*11*) have attempted to use demographic data to examine systematically suicide rates in relation to age, *by sex*. They briefly examined suicide rates for 18 countries by age and sex for 1965 and reported that suicide rates were uniformly higher for males than for females. They also found a general increase with age in suicide rates for males, but the age pattern in suicide rates for females was more inconsistent. Kramer and his co-workers (*11*) also examined the change in suicide rates by age and sex for the same 18 countries from 1955 to 1965. They found that in 12 of the 18 countries, male suicide rates had increased among the age groups under 45 and decreased, or showed small increases, among the age group 45 and over. Also, among the countries showing an increase in suicide rates for women, the most substantial rise in age-specific rates occurred at ages under 45. Thus, the general rise in suicide rates which occurred worldwide from 1955 to 1965 was mainly due to increases in suicide rates at younger ages. Thus, existing studies of suicide need to be augmented by studies that keep suicide in a comparative framework and that carefully differentiate between suicide rates with age for males and those for females.

This paper will examine three topics related to the epidemiology of the age pattern in suicide.

1. The sex difference in the age pattern of suicide will be documented using official statistics both cross-nationally and for the United States.
2. Problems to be overcome in studies of suicide will be discussed.
3. Research and policy issues related to the age pattern in suicide will be presented.

Sex Differences in the Age Pattern of Suicide

In this paper, suicide is defined as death that is judged by the person signing the death certificate as having been self-inflicted. But how valid are the official statistics on suicide? Error can result from (1) varying definitions of suicide, (2) varying criteria for classifying deaths as suicide, (3) varying prevalence of attempts to hide suicides. The crucial question for this paper is whether there is an age or sex bias in the reporting of suicides. It could be, for example, that the decline in position experienced by the elderly (*12*) leads to a decrease in reluctance to classify deaths as suicide. It could also be argued that older people with no children living at home would be more apt to be reported as suicides. However, the facts of the matter are that male suicide rates increase steadily across the entire age range and that female suicide rates are highest precisely when they are most likely to have children still living at home. There can be no doubt that reported suicide rates underestimate "real" suicide rates (*13*), but there is no evidence to indicate a bias that would seriously influence the outcome of the analysis presented in this paper. However, there are strong indications that data on suicides among blacks in the United States are too unreliable to be used for analysis (*3*). Accordingly, the analysis of U.S. data will be confined to the white population.

Three sets of data will be used to examine sex differences in the age pattern of suicide. The first data set consists of official statistics from the World Health Organization. The data on suicide rates were taken from a 1968 special report of the World Health Organization (*14*) which contains suicide rates by age and sex for 25 countries from 1955 to 1966. However, as a control on the quality of cause of death data, five countries—Belgium, France, Greece, Italy, and Venezuela—were excluded since they showed more than 400 deaths per 100,000 from ill-defined or unclassified causes in 1966 (*15*). Data for age-specific death rates from chronic diseases were also taken from this publication (*15*). The second data set consists of cross-sectional data on suicide in the United States compiled by the National Center for Health Statistics for the period spanning 1955 to 1968. The third data set consists of cohort data on suicides tabulated from vital statistics reports spanning 1920 to 1965.

Turning to the cross-national data, tables III–1 and III–2 and figure III–1 show the correlation between age and suicide rates, by sex, for 20 countries. For males there is a near-universal high, positive correlation between age and suicide rates. Japan and Hungary show linear correlations that are depressed slightly by extremely high suicide rates in the oldest age category. Only Poland shows a modest correlation. The correlation data show that despite a wide variety of initial levels of suicide rates (intercepts), the age slopes in suicide rates for males are remarkably consistent from country to country. *Culture thus appears to have an impact on the level of reported suicide among males within various countries, but very little impact on the age trend.*

For females, on the other hand, the situation is very different. Ten of the countries show a correlation of suicide rate with age of 0.80 or more. Seven countries show a moderate correlation between age and suicide rate (0.38 to 0.70), and three show little correlation (0.19 to 0.24). It is noteworthy that females in the German-speaking countries—Federal Republic of Germany, Austria, and Switzerland—show a consistently high correlation between age and suicide rate, while those in the English-speaking countries—Canada, United States, Northern Ireland, Scotland, Australia, and New Zealand—tend toward a moderate to low correlation, England and Wales being the only exception.

Male suicide rates are higher than female suicide rates at all ages in all countries for which there are adequate data. This same result was reported by Kramer et al. (*11*). As shown in tables III–1 and III–2 and in figure III–1 the mean correlation between age and suicide rates for males was 0.89 and for females 0.68.

Tables III–1 and III–2 also show that there is only a modest increase with age in the cross-national standardized variance within age categories, but for both sexes there is a substantial increase in the 75 and over age category. Thus, overall the relationship between age and suicide is linear. The primary departures from linearity occur in the oldest age category because of very high suicide rates after age 75 in Hungary and Japan for both sexes and in the Netherlands among males.

It has been argued often that suicide is related to chronic diseases (*3, 4, 6, 7, 16, 17, 18*), but the evidence has been based primarily on

Table III–1.

Suicide rates by age, variability in suicide rates by age, and correlation of age and suicide rates for males in selected countries, 1963–66

Country	Age							Correlation of age with suicide rate
	15–24	25–34	35–44	45–54	55–64	65–74	75 and over	
Canada	9. 0	14. 6	18. 9	24. 7	29. 7	24. 8	24. 4	0. 84
United States	9. 3	7. 0	22. 0	29. 6	36. 7	37. 4	49. 2	. 99
England and Wales	6. 6	11. 2	15. 2	19. 5	27. 2	27. 6	34. 6	. 99
North Ireland	3. 3	8. 6	8. 0	14. 0	16. 5	12. 0	22. 6	. 89
Scotland	4. 5	8. 8	14. 9	17. 6	22. 6	18. 7	20. 3	. 89
Portugal	6. 4	10. 9	17. 8	26. 6	41. 1	52. 4	64. 3	. 99
Denmark	11. 3	23. 5	34. 0	45. 4	47. 8	44. 1	58. 6	. 94
Norway	4. 6	11. 9	15. 5	21. 6	22. 0	19. 2	19. 0	. 80
Sweden	13. 2	25. 0	36. 5	49. 8	48. 9	46. 6	52. 6	. 90
Netherlands	4. 0	6. 5	7. 3	14. 3	19. 6	22. 2	41. 6	. 93
Federal Republic of Germany	19. 0	24. 9	30. 3	45. 2	52. 7	47. 3	63. 6	. 96
Austria	21. 3	30. 8	39. 2	57. 8	64. 5	54. 2	69. 6	. 93
Switzerland	18. 6	23. 3	32. 6	41. 0	50. 3	53. 4	60. 9	. 99
Hungary	30. 4	43. 2	51. 0	61. 6	66. 1	71. 7	141. 7	. 87
Finland	15. 5	34. 9	51. 6	63. 0	67. 8	67. 0	54. 8	. 80
Poland	12. 0	21. 1	27. 2	28. 0	27. 2	22. 7	21. 1	. 42
Australia	10. 7	23. 5	29. 6	36. 6	36. 1	40. 8	39. 3	. 91
New Zealand	5. 7	9. 9	16. 4	28. 1	28. 3	27. 8	26. 6	. 88
Japan	17. 2	22. 7	16. 0	22. 2	36. 0	53. 0	86. 2	. 87
Israel	4. 5	9. 4	9. 8	15. 7	17. 8	16. 8	22. 2	. 96
Mean	11. 4	19. 1	24. 7	33. 1	37. 9	38. 9	48. 7	. 89
Variance	48. 78	93. 45	164. 15	242. 05	253. 96	291. 80	825. 36	—
Standardized variance	4. 28	4. 89	6. 65	8. 31	6. 70	7. 68	16. 95	—

Table III-2.

Suicide rates by age, variability in suicide rates by age, and correlation of age and suicide rates for females in selected countries, 1963–66

Country	Age							Correlation of age with suicide rate
	15–24	25–34	35–44	45–54	55–64	65–74	75 and over	
Canada	2. 4	5. 6	6. 8	9. 8	9. 6	6. 8	3. 5	0. 23
United States	3. 0	7. 3	10. 1	12. 0	10. 8	9. 4	6. 8	. 41
England and Wales	2. 8	7. 0	10. 3	14. 8	18. 4	19. 9	15. 6	. 89
Northern Ireland	2. 1	3. 4	6. 1	7. 6	8. 6	8. 2	3. 2	. 44
Scotland	3. 0	6. 5	8. 6	11. 6	12. 6	10. 7	6. 3	. 50
Portugal	3. 4	3. 5	4. 2	5. 6	7. 0	11. 0	10. 8	. 95
Denmark	5. 7	12. 5	18. 3	25. 7	28. 4	20. 4	20. 4	. 70
Norway	2. 2	2. 0	4. 9	7. 8	7. 0	12. 6	4. 8	. 65
Sweden	6. 0	12. 4	14. 0	19. 5	17. 0	12. 2	8. 9	. 91
Netherlands	1. 4	4. 0	5. 0	9. 5	11. 4	14. 8	13. 9	. 98
Federal Republic of Germany	6. 4	9. 8	14. 7	23. 0	24. 0	25. 5	25. 8	. 95
Austria	6. 0	9. 1	15. 9	21. 3	22. 5	27. 3	28. 6	. 98
Switzerland	6. 0	10. 1	13. 1	14. 8	16. 8	18. 1	17. 7	. 95
Hungary	12. 5	12. 6	17. 0	26. 4	28. 2	37. 5	52. 9	. 96
Finland	4. 9	9. 4	13. 5	19. 2	14. 2	15. 1	6. 2	. 24
Poland	3. 4	3. 3	4. 3	6. 2	6. 2	5. 7	5. 7	. 82
Australia	5. 3	12. 2	17. 0	22. 6	21. 4	18. 3	10. 0	. 38
New Zealand	3. 9	4. 8	6. 8	15. 4	20. 4	14. 6	8. 2	. 57
Japan	14. 0	14. 5	10. 2	13. 8	19. 6	40. 0	66. 8	. 81
Mean	4. 8	7. 7	10. 4	14. 9	16. 0	17. 4	16. 5	. 68
Variance	10. 23	13. 91	21. 35	40. 65	44. 43	82. 65	261. 00	—
Standardized variance	2. 13	1. 81	2. 05	2. 73	2. 78	4. 75	15. 82	—

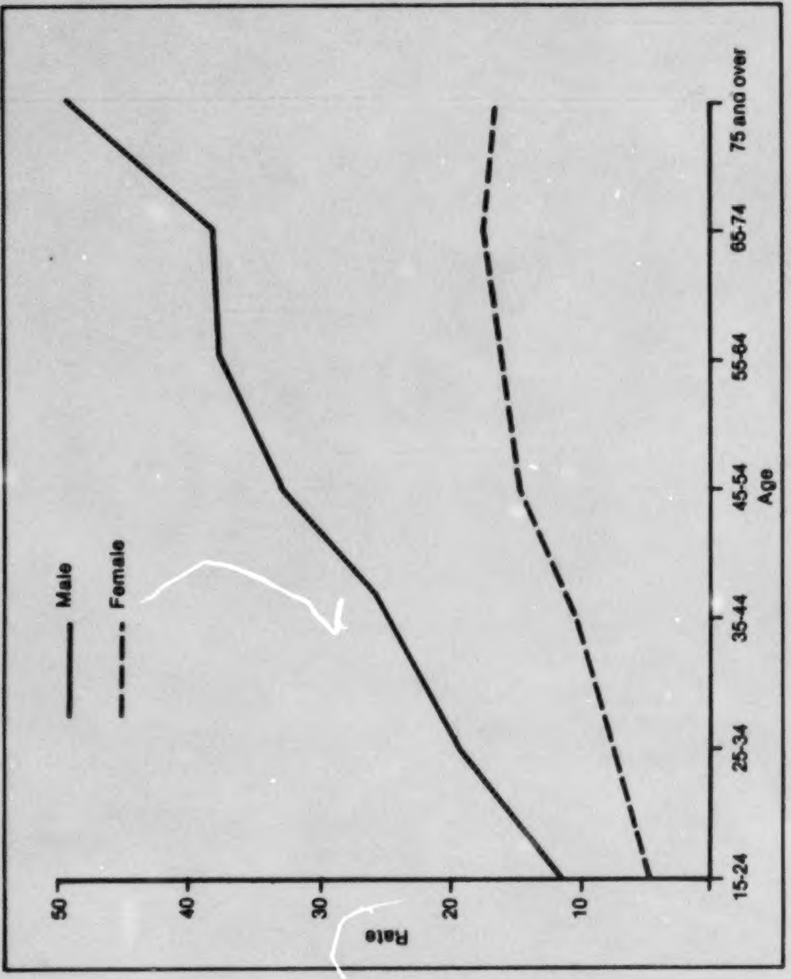

Figure III–1.
Mean suicide rate, by age and sex, for 20 selected countries, 1963–66.

impressions and small numbers of cases. Many studies link suicide with illness through reports from people who are acquainted with the suicide victim. This sort of evidence is somewhat suspect, since physical illness may be seen as a more socially acceptable reason for suicide as compared to other reasons. Tuckman, Youngman and Kriegman (*19*) correlated suicide rates with death rates from 72 diseases for a sample of white males age 20 to 64 in the United States in 1950. They argued that mortality rates are an indication of prevalence of various diseases. They found that suicide rates correlated highest with the

Table III-3.

Regression of selected chronic disease death rates to suicide rates[1] for selected countries[2], by age and sex, 1966

Total			45–54			55–64			65–74			75 and over		
Cause	Mult. R	R^2	Cause	Mult. R	R^2	Cause	Mult. R	R^2	Cause	Mult. R	R^2	Cause	Mult. R	R^2
							Males							
A 46	0. 45	0. 20	A 82	0. 47	0. 22	A 82	0. 41	0. 17	A 46	0. 45	0. 20	A 46	0. 61	0. 37
A 83	. 50	. 25	A 63	. 50	. 25	A 70	. 46	. 21	A 63	. 63	. 40	A 83	. 80	. 64
A 47	. 64	. 41	A 50	. 54	. 29	A 63	. 52	. 27	A 83	. 72	. 51	A 85	. 82	. 68
A 112	. 67	. 45	A 83	. 57	. 32	A 47	. 56	. 32	A 112	. 78	. 61	A 63	. 84	. 71
A 85	. 68	. 46	A 85	. 61	. 37	A 83	. 47	. 34	A 47	. 84	. 70	A 82	. 85	. 73
A 63	. 69	. 48	A 70	. 64	. 41	A 112	. 60	. 36	A 81	. 86	. 74	A 81	. 86	. 74
A 50	. 70	. 49	A 81	. 67	. 45	A 81	. 61	. 37	A 85	. 87	. 75	A 47	. 89	. 79
A 70	. 71	. 50				A 50	. 67	. 45	A 82	. 87	. 76	A 70	. 89	. 81
							Females							
A 46	0. 29	0. 08	A 81	0. 18	0. 03	A 50	0. 24	0. 06	A 46	0. 57	0. 33	A 46	0. 56	0. 31
A 81	. 53	. 28	A 85	. 27	. 01	A 85	. 33	. 11	A 85	. 64	. 41	A 63	. 66	. 44
A 70	. 57	. 33	A 63	. 34	. 12	A 82	. 41	. 17	A 50	. 66	. 44	A 83	. 72	. 52
A 50	. 63	. 40	A 70	. 44	. 19	A 83	. 45	. 20	A 47	. 71	. 50	A 70	. 84	. 70
A 83	. 66	. 43	A 46	. 52	. 27	A 63	. 48	. 23	A 63	. 74	. 54	A 81	. 85	. 72
A 85	. 70	. 49	A 50	. 59	. 24				A 81	. 79	. 62			
			A 47	. 63	. 39									

A 46	Cancer, stomach
A 47	Cancer, intestine
A 50	Cancer, lung
A 63	Diabetes
A 70	Stroke
A 81	Degenerative heart disease
A 82	Other heart disease
A 83	Hypertension with heart disease
A 85	Artery disease
A 112	Hyperplasia of the prostate

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[1] Suicides rates based on the 1962–66 average (see tables III-1 and III-2).

[2] For a list of countries see table III-1.

death rate from all causes (0.88), followed closely by tuberculosis, alcoholism, ulcer of the stomach, and all forms of cancer. Their units of analysis were 26 occupational groups. However, their study did not take age into account or explicitly examine age as a variable.

In the present study, a stepwise multiple regression analysis was performed on cause of death data from the World Health Organization to assess the extent to which the death rates from 10 chronic diseases would predict suicide rates for the 20 countries shown in tables III–1 and III–2. The analysis, presented in table III–3, begins with the age category 45 to 54 because chronic disease death rates tend to be insignificant before that age.

Table III–3 shows that below age 65, chronic disease death rates predict only a modest proportion of the cross-national variance in suicide rates (0.23 to 0.45). After age 65, however, a composite of chronic disease death rates produces a high R-square *for both sexes.*

Chronic disease death rates produce a regression equation which predicts suicide rates better for males than for females, regardless of age. As age increases, however, the sex differential declines. At age 75 and over there is little sex difference in the ability of chronic disease death rates to predict suicide rates. These data suggest that the lower average correlation between age and suicide rates among females may be, in part, related to a lower prevalence of terminal chronic diseases among women under age 75.

Of course, these sort of data do not show that having chronic diseases causes people to commit suicide. What it does show is that those countries with relatively high death rates from chronic diseases also tend to have high suicide rates, and this is true particularly after age 65 for both sexes. Some diseases have high illness rates, but low mortality rates, while others have low illness rates, but high mortality rates. However, for any country, the higher the mortality rate for a given disease, the higher the prevalence of that disease, particularly terminal cases. Thus, the correlation between death rates from chronic diseases and from suicides is a strong indication that longitudinal studies of illness and suicide might prove fruitful.

Table III–4 shows the simple correlations of the various chronic disease death rates with suicide rates by age and sex. In conjunction with table III–3, these data show that while suicide rates generally can be predicted from a composite of chronic disease death rates, the most important diseases in producing that prediction change with age. For males, there is a decline in the importance of heart disease and an increase in the importance of cancer of the stomach, and of hypertension and stroke. For females, there is an increase with increased age of the cohort of the importance of cancer of the stomach and stroke.

These data suggest various ways that chronic disease may be related to suicide. For both men and women at age 45 to 54, suicide rates are generally unrelated to particular chronic diseases, with the single exception of nondegenerative heart disease among the men. At ages 55 to 65 suicide rates begin to be related moderately to death rates from several particular chronic diseases among men, but not among women. This is probably related to the overall lower incidence of terminal chronic disease among women at this age.

The rise in importance of cancer of the stomach and of hypertensive heart disease and stroke after age 65 relates to two presumed "causes" of suicide. It is generally assumed that people with hopeless and painful prognoses are attracted to suicide. The link between death rates from cancer of the stomach and suicide rates suggest that this hypothesis applies most strongly after age 65. It is also often assumed that suicides do not have their wits about them, that the usual impulse controls have failed. The correlation of high rates of death from hypertension and stroke, diseases which in their later stages tend to produce

Table III–4.

Correlations of death rates from selected chronic diseases with suicide rates for selected countries,[1] by age and sex, 1966

Cause	Total	45–54	55–64	65–74	75 and over
MALES					
A 46	0. 45	0. 06	0. 06	0. 45	0. 61
A 47	. 17	–. 16	. 37	–. 30	–. 35
A 50	. 13	–. 06	. 34	–. 09	–. 08
A 63	. 20	. 27	. 02	. 32	–. 14
A 70	. 41	–. 06	. 39	. 40	. 43
A 81	. 14	–. 01	–. 26	–. 26	–. 29
A 82	. 15	. 47	. 41	. 04	–. 22
A 83	. 42	. 08	. 40	. 36	. 46
A 85	. 22	. 25	. 33	–. 14	. 12
A 112	. 27	–. 16	. 18	. 39	. 08
FEMALES					
A 46	0. 29	–0. 07	0. 03	0. 57	0. 56
A 47	–. 11	. 03	. 15	–. 40	–. 38
A 50	. 21	–. 03	. 24	. 15	. 30
A 63	–. 07	–. 07	. 03	–. 09	–. 28
A 70	. 16	–. 04	. 03	. 51	. 45
A 81	–. 08	. 18	. 05	–. 35	–. 44
A 82	–. 08	–. 10	–. 01	–. 16	–. 22
A 83	. 09	–. 05	–. 15	. 05	. 16
A 85	. 04	–. 15	–. 20	–. 13	. 05

A 46	Cancer, stomach
A 47	Cancer, intestine
A 50	Cancer, lung
A 63	Diabetes
A 70	Stroke
A 81	Degenerative heart disease
A 82	Other heart disease
A 83	Hypertension with heart disease
A 85	Artery disease
A 112	Hyperplasia of the prostate

[1] For a listing of countries, see table III–1.

marked susceptibility to "depression," would seem to indicate that there is a possible organic basis for this "emotional" factor after age 65, but not before. These analyses are purely speculation and suggestion. However, they do indicate some promising directions that could be used to explore relations between health and suicide.

To relate the age pattern in suicide to the degree of urbanization and industrialization, the percentage of the population classified as urban and per capita gross national product were taken as indicators. Tables III–5 and III–6 show the relationships among the correlation of age and suicide rate, by sex, and the percentage of the population residing in urban areas, and per capita GNP for 20 countries. For males, there is a mild positive association between the degree of urbani-

Table III–5.

Correlation between age and suicide rate for males, percent urban and per capita gross national product, for selected countries, 1966

Country	Correlation of age with suicide rate [1] (1)	Percent urban [2] (2)	Per capita GNP [3] (3)
Portugal	0.99		380
Switzerland	.99	54.6 [4]	2,250
England and Wales	.99	79.0	1,620
United States	.99	73.5	3,520
Federal Republic of Germany	.96	38.4	1,220
Israel	.96	81.8	1,160
Denmark	.94	46.2	1,830
Austria	.93	51.9	1,150
Netherlands	.93	78.4	1,420
Australia	.91	83.3	1,840
Sweden	.90	77.4 [5]	2,270
Scotland	.89	75.7	1,620
Northern Ireland	.89	53.2	850
New Zealand	.88	68.3	1,930
Japan	.87	68.1 [6]	860
Hungary	.87	44.9	800
Canada	.84	73.6	2,240
Finland	.80	45.3	1,600
Norway	.80	40.8	1,710
Poland	.42	48.9 [6]	730

$r_{1.2} = .35$ $r_{1.3} = .05$

[1] From table III–1.

[2] From *Demographic Yearbook*, 1971.

[3] From *International Bank for Reconstruction and Development, 1966.*

[4] 1970.

[5] 1963.

[6] 1965.

zation and the correlation between age and suicide rates. There is no relationship between per capita GNP and the correlation between age and suicide rate. For females, there is a mild *negative* association between degree of urbanization and the correlation between age and suicide rates. There is a moderately high *negative* relationship (−0.71) between per capita GNP and the correlation between age and suicide rates.

From these data, it appears that the *age pattern* in suicide rates is related to overall economic factors only among females. This relationship needs further scrutiny. A likely place to look would be the relationship between per capita GNP and the prevalence of terminal chronic diseases, especially at the older ages. If per capita GNP depresses

Table III–6.

Correlation between age and suicide rate for females, percent urban and per capita gross national product, for selected countries, 1966

Country	Correlation of age with suicide rate [1] (1)	Percent urban [2] (2)	Per capita GNP [3] (3)
Austria	0. 98	51. 9	1, 150
Netherlands	. 98	78. 4	1, 420
Hungary	. 96	44. 9	800
Portugal	. 95	–	380
Federal Republic of Germany	. 95	38. 4	1, 220
Switzerland	. 95	54. 6 [4]	2, 250
Israel	. 93	81. 8	1, 160
England and Wales	. 89	79. 0	1, 620
Poland	. 82	48. 9 [5]	730
Japan	. 81	68. 1 [4]	860
Denmark	. 70	46. 2	1, 830
Norway	. 65	40. 8	1, 710
New Zealand	. 57	68. 3	1, 930
Scotland	. 50	75. 7	1, 620
Northern Ireland	. 44	68. 3	1, 930
United States	. 41	73. 5	3, 520
Australia	. 38	83. 3	1, 840
Finland	. 24	45. 3	1, 600
Canada	. 23	73. 6	2, 240
Sweden	. 19	77. 4 [6]	2, 270

$r_{1.2} = -0.29$ $r_{1.3} = -0.71$

[1] From table III–1

[2] From *Demographic Yearbook* 1971

[3] From *International Bank for Reconstruction and Development*, 1966.

[4] 1970.

[5] 1963.

[6] 1965.

chronic disease rates at older ages for women, as one might well suspect that it might, then it could destroy the correlation between age and the suicide rates as well.

Urbanization apparently has little impact on the correlation between age and suicide rates for either sex, but what effect it does have is in opposite directions. A high correlation between age and suicide rates tends to be associated with a high degree of urbanization for males, but among females a low degree of urbanization is associated with a high correlation between age and suicide rates. Again, it may be possible in future research to link these data for females to the impact of urbanization on terminal chronic diseases.

These cross-national data have some obvious implications for the study of suicide. The consistent linear relationship between age and suicide rates suggests a possible organic basis to suicide which deserves more attention than this possibility has received in the past. Longitudinal studies of diagnosed terminal chronic disease cases would seem to be a particularly fruitful course for future inquiry.

We will now turn to the U.S. data to see what has been happening to the age pattern in suicide over time. Table III–7 shows that for the white population, there was little change from 1955 to 1966 in the correlation between age and suicide rates among males, while there was a sharp decline in the correlation between age and suicide rates for females. For both men and women, suicide rates have risen in the younger age categories and declined among the older categories. This phenomenon has been attributed to the alienation of youth and to better programs for the elderly. If that is the case, then these conditions must apply worldwide since Kramer, et al. (*11*) observed this same pattern among 18 countries.

Age-specific suicide rates are consistently higher for men than for women. This also was true without exception for the 20 countries in the earlier analysis.

These data say more about what does *not* produce high suicide rates than about what does. For one thing, there is obviously no surge in suicides in the age category in which retirement usually occurs (65 to 74), for either sex. Even if one looks at the 65 to 69 age category, there is, if anything, a slight depression of suicide rates in the 65 to 74 age category in comparison with the regression line. Most women encounter the empty nest between age 45 and age 54. There is no surge in suicide rates associated with this age category. The empty nest does not appear to influence the suicide rates in any significant way. In fact, although ours is a highly age-graded society, there are no discernible sudden surges or declines in suicide rates connected with age. Yet among males, the relation between age and suicide is universal.

In an attempt to pursue the case further, data were compiled on suicide rates of seven 5-year birth cohorts. Beginning with 1920, the seven cohorts were followed through the vital statistics records to 1965. The results are presented in table III–8. From these data, several addi-

Table III–7.
Suicide rates by age and sex and correlation of suicide rates with age, by sex: United States, 1955–1968 (for whites)

	All ages	15–24	25–34	35–44	45–54	55–64	65–74	75 and over	r
MALES									
1968	15.8	10.9	17.1	22.0	27.2	34.0	34.5	43.1	0.992
1967	15.7	10.5	17.2	22.9	27.5	34.4	32.9	42.8	.982
1966	16.1	9.7	17.3	21.6	28.5	36.2	36.8	49.3	.988
1965	16.3	9.4	17.3	22.6	29.1	37.3	37.4	47.4	.990
1964	16.1	9.2	16.9	21.2	29.9	36.0	37.0	49.4	.988
1963	16.5	9.0	16.6	22.4	30.7	37.3	38.3	50.4	.990
1962	16.5	8.5	15.9	21.6	30.9	38.0	38.8	54.1	.986
1961	16.1	7.9	14.9	21.2	31.0	37.4	37.7	51.3	.988
1960	16.5	8.1	14.7	21.1	31.5	37.9	40.4	55.5	.989
1959	16.6	7.6	14.4	20.7	30.8	39.7	46.0	55.8	.998
1958	16.8	7.2	14.2	21.3	31.9	39.8	46.4	55.5	.999
1957	15.4	6.3	12.7	19.3	28.4	36.1	43.8	53.9	.998
1956	15.7	6.2	12.7	18.4	28.2	39.4	45.6	54.6	.996
1955	16.0	6.1	12.4	18.8	29.7	40.6	44.5	53.1	.995
FEMALES									
1968	5.9	3.4	7.2	10.7	12.4	10.8	7.7	6.0	0.218
1967	6.1	3.5	7.6	10.7	12.1	11.5	9.4	6.4	.327
1966	5.9	3.1	7.6	10.4	12.0	10.8	9.3	6.6	.363
1965	6.1	3.0	7.4	10.1	12.7	11.4	9.4	7.5	.454
1964	5.6	2.8	6.9	10.1	11.6	10.2	9.8	6.0	.387
1963	5.8	3.1	7.2	9.9	11.9	10.9	9.1	7.0	.429
1962	5.4	2.9	6.8	8.8	11.5	10.4	8.2	7.3	.487
1961	4.9	2.3	5.8	7.8	9.9	9.7	8.5	6.8	.609
1960	4.9	2.2	5.5	7.7	10.1	10.1	8.5	8.6	.754
1959	4.7	2.1	5.5	7.0	8.7	10.0	10.0	6.7	.710
1958	4.7	2.3	5.6	6.7	9.6	9.4	9.4	6.4	.659
1957	4.3	1.8	4.7	6.6	7.9	9.6	8.3	5.7	.667
1956	4.4	1.9	4.5	6.3	8.9	9.9	9.3	6.2	.697
1955	4.6	2.0	4.6	6.1	9.5	9.8	9.2	7.8	.801

tional facts emerge. First, the age-specific suicide rates have generally increased at younger ages and declined at older ages for successive cohorts for both sexes. Second, the relation between age, sex, and suicide rates observed in table III–7 for cross-sectional data holds in table III–8 for cohort sequential data. This means, of course, that we can have more faith in our generalization that age bears a stronger relationship to suicide among males than among females. We also now know from table III–8 that the increase in suicide rates at younger ages began in the 1930's as did the decline in suicide rates at older ages. Thus, the roots of the causes for these patterns must be searched out in the social conditions of the 1930's and not the 1960's as has been widely suggested.

The cohort sequential data in table III–8 also allow us to examine changes in age-specific suicide rates both in the context of the suicide history of specific birth cohorts and in the context of various historical periods. Only males were used because the variability among females was too low for meaningful analysis. Figure III–8 shows that for males an increment in suicide rates with age is generally present for each measurement point from 1920 to 1965. The increase in suicide rates with age *within* cohorts is also obvious. Not so obvious, is the impact of contemporaneous events on the *range* of variation of suicide rates by age. In 1925, the range in suicide rates for the seven cohorts was 10.7 to 41.0, or 30.3 suicides per 100,000. In 1930 the spread was 19.2 to 67.4, or 48.2 per 100,000. By 1940, the spread was back down to 29.2, and it stayed in the 1920's through 1955, the last date for which we have information on all seven cohorts. Thus, the decade of the 1930's produced an expansion of range of variation among the seven cohorts in suicide rates by age which was atypical either before or after that decade. Another not so obvious point from figure III–2 is what appears to be a leveling off of suicide rates for the younger cohorts since 1955.

From the data presented in this paper, it appears that the direct relationship between age and suicide rates among males is not the product of any particular culture or of any ongoing events in the society. These factors influence the general level of reported suicides, but not the relationship between age and suicide rates. Neither is it a statistical artifact since the relationship holds over time in cross-national, cohort sequential, and cross-sectional data.

Thus, aging represents a consistent factor in suicide among men but not among women. Later in this paper some suggestions will be given for research to learn *why* this sex difference occurs. Certainly, the steady increase in suicide rates for males across the entire life span indicates that this factor must be given consideration in any overall approach to the epidemiology of aging.

Future Research

Research on suicide and aging faces even more problems than do studies of suicide in general. The most difficult problem is to differentiate the following categories: (1) Persons who died of other causes, (2) persons who overtly acted to kill themselves, (3) persons who by not acting (eating, taking medication, etc.) willfully died, and (4) persons who by ill-advised action or inaction *unintentionally* killed themselves. Only categories 2 and 3 are "true" suicides. At the present time we have few clues as to the magnitude of categories 2, 3, and 4.

Table III-8.

Suicide rates by age and sex for selected 5-year birth cohorts: United States, 1920–65

	20–24	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60–64	65–69	70–74	75–79	80–84
WHITE MALES													
1965									38.9	37.8	41.2	47.8	52.9
1960								39.9	40.6	38.5	46.7	53.1	61.2
1955							36.1	41.7	44.0	45.0	46.3	53.1	57.8
1950						31.2	37.4	43.5	48.7	52.3	54.5	60.1	
1945					24.2	25.8	32.6	36.6	40.9	41.9	48.7		
1940				28.3	32.0	40.7	47.8	57.6	60.1	57.5			
1935			22.2	25.6	32.7	44.1	50.2	57.4	59.8				
1930		19.2	24.1	31.3	41.7	50.3	62.0	67.4					
1925	10.7	14.1	17.6	23.2	28.0	35.6	41.0						
1920	10.6	14.0	17.6	20.4	25.5	27.5							
WHITE FEMALES													
1965									10.9	11.2	8.6	8.1	7.6
1960								10.8	10.9	8.5	9.3	9.9	7.9
1955							11.1	10.9	9.9	10.2	8.3	8.4	8.2
1950						9.9	11.1	10.4	10.9	11.1	10.0	8.7	
1945					9.8	11.4	12.5	12.0	10.6	11.0	10.8		
1940				10.9	12.1	14.1	13.8	13.2	13.0	13.6			
1935			10.5	10.7	11.1	13.0	13.1	13.4	14.4				
1930		9.5	10.0	10.7	12.2	12.7	13.8	14.8					
1925	6.3	7.8	7.9	9.2	9.8	11.0	12.3						
1920	6.9	8.1	8.3	9.5	10.5	12.1							

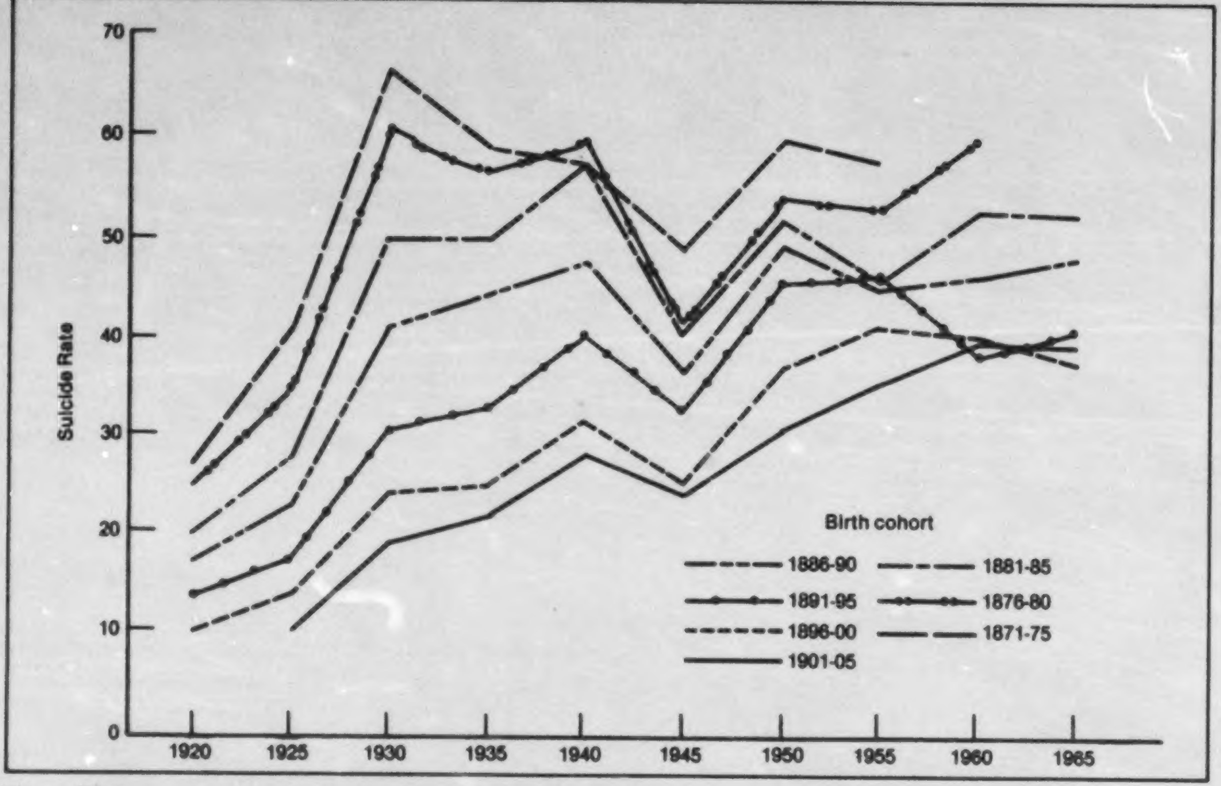

Figure III-2.
Male suicide rates for various birth cohorts: United States, 1920–1965, white population.

More widespread use of psychological autopsies would certainly help data quality problems a great deal. Longitudinal data sets could be analyzed to help establish temporal sequence in relating health problems to suicide.

The main failing of existing studies of suicide is their unidisciplinary focus. Studies generally stress either psychological factors or social factors. As a result, no studies have been done that address the *relative* influence of physical, social, and psychological factors in the etiology of suicide.

Models that emphasize multiple causes and multidisciplinary methodology and theory will be necessary to unravel the various unknowns that pervade the literature on suicide. On the physical side it would be useful to know how biochemical changes in neurotransmission influence impulse control and suicide. We also need to know how various diseases relate to eventual suicide. Is suicide among older men related to higher incidences of functional disability? It would also be useful to know the role that medication plays in suicide. Are suicides more often taking particular medications compared to nonsuicides?

On the social side, we need to get away from general causal models. Anomie, egoism, altruism, or fatalism may indeed be related to suicide among older people, but *concrete situations* need to be identified that predispose people to suicide whether or not these situations operate through the traditional Durkheimian variables. In explaining the sex difference in the age-pattern of suicide, it might be useful to consider whether widowers who commit suicide become isolates prior to suicide. Is suicide among older men related to a reluctance to accept physical dependency? Is suicide among older men related to a tendency to mismanage medication? These are but a few of the specific social factors that could be considered.

World view is a sociocultural factor worth considering in suicide research. Certainly a person who believes that physical death is meaningless is not under the same constraint against suicide that controls someone who believes that death represents the end of everything.

Psychological factors in suicide research need to be expanded to include more interaction with physical and social factors as paths in arriving at states such as suicidal depression.

From a policy point of view, there are two issues that are central to research on suicide and aging. The first is to get estimates of the "true" rates of suicide that include both active and passive suicides. The second is to place suicide into a context of social escape. To the extent that suicide represents escape from a low-quality life, it needs to be included in an overall framework that includes alcohol and drug abuse, the two other main escape routes in American society. It could very well be that self-destructive behavior is a much more prevalent reaction to a low-quality life than looking at suicide alone would indicate. Knowledge on these issues will tell us more about whether research on suicide and aging should get a high priority.

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Discussion

Costa: I would like to address my comments to table III–7 showing the age and sex differences in relation to suicide rates and possibly suggesting the relationship to the midlife crises.

If there is a midlife crises that we hear a lot about today, it certainly does not seem to be manifest in the data for the males where there is a beautifully linear relationship with age. I do not want to make a mountain out of a couple of percentage points difference but it looks like the interval 45 to 54 is a rather nice mode for females and that is close to that area.

Atchley: That is true in the older cohorts but you will not find it in the latest one. If you look at table III–7 at the top of the diagonal, you will see a different pattern. You do have that kind of little hump in the middle for the oldest cohort at the bottom diagonal, but it disappears as you go up in the cohort structure. That leads me to believe that it is a passing factor. I think that you would have to make a mountain out of a small percentage point in order to draw your conclusions about midlife crises and suicide from these data.

Costa: The safest interpretation would be that, if there is a midlife crisis, it does not express itself in differential suicide rates of those nations.

Waldron: It could be proposed that women are more successful at using a suicide attempt as a plea for help, kind of a last ditch plea for help, and men are unable to plea for help in this way and instead actually kill themselves.

Siegel: But the data are confounded by the fact that you have failed attempts which succeed among the women.

Cohen: You also have bias of another kind which is that women are more likely to still be connected to those who will not report their deaths as suicides or who will try to disguise them, in contrast to isolated men whose deaths will more likely be reported as suicides.

Siegel: That is an important question.

Atchley: Which brings us back to the idea, to what extent are suicides accidents? When you have advanced technology, there may not be good data on the types of methods that are chosen. For example, if what is reported as suicide in older men is really the result of depression brought on by medication, then the death may actually be the result of an unintentional overdose. There is some work that is being done with psychological autopsy materials to try to differentiate suicides from accidents that appear to be suicides, but not really anything that can answer your question.

Adelman: Is it true that women are less inclined to self destruction or am I to consider that they are less successful in accomplishing this?

Atchley: Well, the literature that I have read does indicate that males, when they do choose to commit suicide, choose more foolproof methods.

Gruenberg: Could you explain that more? Are you making distinctions between attempted suicide and suicide?

Atchley: The question was, "Is it that females are just less inclined to be suicidal or is it that they are just less effective in carrying out their intent?" I really cannot answer the question, although the statistics show that there are more attempts among females, but there are more successes among males.

Gruenberg: So why do you view these attempts as real serious attempts? I mean attempted suicide is a different phenomenom altogether than suicide.

Atchley: Yes, I know. The data on attempts are not classified as serious.

Palmore: It seems to me your figure III–2 is a classic illustration of the problem of disentangling age, period, and cohort effects. Now specifically, you said that there is a cross sectional difference at all age periods with which I will go along, generally, but I thought you also said that there was a longitudinal increase with age in each cohort. Now, I do not see that for the top three, certainly, or even four cohorts. It looks like beyond 1935 and 1940, it just bounces up and down.

Atchley: What happened was that the depression expanded the range but after the depression it came back down to the former trend.

Palmore: You could say the opposite, that for the top three cohorts there was no increase in suicide after 1930 or 1935.

Atchley: Yes, but it makes more sense given the whole pattern of data to say that if the period effect reduced the difference at that point, that the general pattern is one of linear increase.

Sacher: Nietsche once said that the thought of suicide was the only thing that got him through a bad night. To what extent is the contemplation of suicide related to the actual completion of the act?

Atchley: I do not know. I have not seen any research to identify people who have contemplated suicide.

Kannel: I am wondering, having done a fair number of death certifications in my lifetime, how large a role variation in death certification practices play in determining what percentage of actual suicides get recorded. Does this vary by age, by sex, and by country?

Cohen: There is pretty good research on this and it varies by all of those and by area, literally county to county, according to the practices of the coroner and the investigational ability available, prevailing cultural norms, and other factors.

Kannel: I would think so, and by ethnic origin and religious group.

Cohen: And there is a huge sex difference. I cannot point that out too strongly. Women are much less likely to be classified as suicides.

Atchley: The sex difference operates in the opposite direction, though, in the pattern that I showed here.

Cohen: No, if you assume that there is underreporting for women, the differential rates between women and men would increase. I think that is happening over time; that the differential is changing.

Atchley: Yes, the differential is changing; that is true, but it is interesting that the suicide rates for females are down.

Brody: Since alcoholism is very heavily sex dependent, how would your pattern be influenced if you subtracted out the alcoholics?

Atchley: Among older people who attempted suicide were those who had a high incidence of alcohol and drug problems prior to their attempt. As far as the exact proportions and probabilities, I do not think that we have the information to gauge that at this point.

Brody: There is a fair amount of data on this and there is a very strong difference between the sexes for alcohol. What I am suggesting is that it is an independent variable with the same sex pattern. Perhaps clarification of that would reduce the differences among the majority of suicides.

Cohen: I think that part of the problem is the confounding one between alcohol as an indicator of a chronic behavioral condition and alcohol use immediately prior to and as a precipitant to the suicidal act.

Siegel: Some years ago at the planning meeting for the establishment of NICHD, I made the comment that one of the most important goals of the agency should be that every child should be born wanted and well insofar as possible. Now, I think that NIA, particularly, ought to get into the research on the bioethics of dying as a special field and should consider the whole area of the "right to die," suicide education and planning, and the allocation of scarce resources for treatment of serious illness. That is to say, we should explore the proposition that everyone has the right to die as well as to live.

Atchley: There was an interesting excerpt from a death note, a suicide note of a woman author who said, "what I am doing right now is evaluating my life. Seeing no future of anything but misery, I am exercising my most fundamental right, the right to end my life."

Siegel: After I made a public statement about the problem of allocating scarce resources in the health area, I received a disturbing but very thoughtful anonymous letter from a widow giving some reflections on the matter. She said that she has spent many miserable years as a widow and wished that she had had the "guts" to commit suicide when "it" first happened. I do not offer this as an example of what we ought to encourage people to do, but rather as an illustration of the kinds of pressures and concerns that affect our older population and of the fact that many would claim suicide as a right under certain conditions. Older people may perceive this issue differently from younger persons.

Sex Differences in Longevity

Ingrid Waldron, Ph. D.*

University of Pennsylvania
Leidy Lab/G7
Department of Biology
Philadelphia, PA 19104

Why do women live longer than men? An analysis of this question for the contemporary United States has demonstrated that the sex difference in mortality is due primarily to coronary heart disease and a number of behavioral causes of death (*1*, *2*). Because coronary heart disease plays such a major role, the causes of sex differences in coronary heart disease are probed in greater depth in this paper. Finally, additional insight into the causes of sex differences in mortality is provided by analyses of cross-cultural and historical variation in sex mortality ratios.

Mortality in the Contemporary United States

In the United States, the life expectancy of a baby boy at birth is 68 years, 8 years shorter than that of a baby girl (*1*). Which causes of death are responsible for this sex difference in longevity? The major causes of death for which male mortality is at least twice as high as female mortality are accidents, suicide, cirrhosis of the liver, lung cancer, emphysema and coronary heart disease (table III–9). For each of these causes of death, a person's risk is strongly influenced by his or her behavior. In each case, behaviors which are more common among males contribute to their elevated mortality (*1*, *2*).

Specifically, cancers of the respiratory system and emphysema and other bronchopulmonary diseases are more common in men primarily because men smoke cigarettes more (*1*). In addition, approximately one-fourth of men's lung cancer deaths may be due to exposure to industrial carcinogens on the job. Cirrhosis of the liver and fatal motor vehicle accidents are more common in men in part because men consume more alcohol than women (*2*). Similarly, men appear to have less safe driving habits. Other types of accidents which are more common for men include principally work accidents, drownings and gun accidents (*2*). Men have higher suicide rates than women in part because they use guns more often (as opposed to poisons, which are often less lethal) and probably also because they experience more stresses related to unemployment. Taken together these causes of death with major behavioral components are responsible for a third of men's excess mortality over that of women (*1*, *2*).

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Table III–9.
Major causes of higher mortality in men

Sex mortality ratio [1]	Cause of death	Male death rate [2]	Female death rate [3]
5. 9	Malignant neoplasm of respiratory system, not specified as secondary	50. 1	8. 5
4. 9	Other bronchopulmonic disease (71 percent emphysema)	24. 4	5. 0
2. 8	Motor vehicle accidents	39. 4	14. 2
2. 7	Suicide	15. 7	5. 8
2. 4	Other accidents	41. 1	17. 4
2. 0	Cirrhosis of liver	18. 5	9. 1
2. 0	Arteriosclerotic heart disease, including coronary disease	357. 0	175. 6
1. 6	All causes	1, 081. 7	657. 0

This table lists all causes of death which had a sex mortality ratio of 2 or more and were responsible for at least 1 percent of all deaths in the United States in 1967. These causes of death were responsible for three-quarters of the sex differential in mortality. (See reference 1 reprinted with permission from the Journal of Human Stress).

[1] The sex mortality ratio is the ratio of male to female death rates.

[2] Deaths per 100,000 population per year.

[3] Female death rates have been age-adjusted using the age-specific death rates for females and the age distribution for males to calculate the death rate which would be expected for a population of females that had the same age distribution as the male population. Thus the male and female death rates are directly comparable and are not affected by the higher proportion of females at older ages.

Forty percent of the sex difference in mortality is due to arteriosclerotic heart disease, which is twice as high for men as for women. One reason why men suffer more coronary heart disease than women is that men smoke more (*1*). In addition, men display the hard-driving, rushed, competitive type A or coronary-prone behavior pattern more often than women (*3*), and this behavior pattern is associated with a substantially elevated risk of coronary heart disease.

Overall, then, a third of the sex differential in mortality in the United States has been found to be due to accidents, suicide, cirrhosis of the liver, lung cancer and emphysema, each of which has major behavioral components. Cigarette smoking and the hard-driving coronary-prone behavior pattern contribute to a higher rate of coronary heart disease among men, and coronary heart disease is responsible for 40 percent of the sex difference in mortality (*1*).

Causes of Sex Difference in CHD

One cause of women's lower risk of coronary heart disease may be a protective effect of female sex hormones. Results have been inconsistent, but a majority of the available studies report that after oophorectomy or natural menopause women have an increased risk of angina pectoris, myocardial infarction or clinical coronary heart disease (*1, 4*). Current data do not permit a quantitative estimate of the strength of this effect, but the incidence of coronary heart disease in postmenopausal women appears to be intermediate between the incidence in men and in premenopausal women of the same age (*4, 5*). These data must be interpreted with caution because of substantial methodological problems, such as the failure to distinguish women who have had only their uterus removed from those who have had both ovaries removed (*1, 4*). After oophorectomy, replacement therapy with estrogens may reduce the risk of clinical coronary heart disease, although again the findings have not been consistent (*1*).

Estrogen therapy in menstruating women and in men usually produces an *increased* risk of coronary heart disease. This result can be understood in light of the multiple effects of estrogens, including enhanced thrombotic processes (*1*). Studies of estrogen levels in young men who have suffered a myocardial infarction have produced inconsistent results. One study reports elevated levels of estrogens and a variety of bodily symptoms associated with elevated estrogen levels (*6*). In contrast, other workers report no elevation in estrogen levels (*7*) and an inconsistent tendency to slightly more masculine body build (*8*) for men who have experienced a myocardial infarction. Thus the available evidence suggests that estrogens do not reduce the risk of coronary heart disease in men, although they may do so in women.

The mechanisms by which estrogens may decrease the risk of coronary heart disease in women remain unclear. Studies of atherosclerosis following oophorectomy have yielded highly variable results which suggest that oophorectomy may lead to increased coronary atherosclerosis, though possibly only with a lag of a decade or more (*9–12*). The decrease in sex hormone levels at menopause seems to be associated with an increase in serum cholesterol levels, but not in other standard risk factors for coronary heart disease (*13*). A protective effect of female sex hormones would appear to involve primarily physiological changes not reflected in the standard risk factors (*4, 14, 15*) In summary, endogenous female hormones appear to reduce the risk of coronary heart disease, but the magnitude of the effect and the mechanism remain unclear.

A survey of sex differences in arteriosclerotic heart disease in different countries supports the hypothesis that the sex differences may be due in part to hereditary factors such as sex hormones; men's death rates for arteriosclerotic heart disease are higher than women's in almost every country studied (*16, 17*). On the other hand, international data also indicate that cultural factors make an important contribution to the sex difference in arteriosclerotic heart disease. The sex mortality ratio varies widely, from 0.9 in Honduras and 1.0 in Guatamala to 2.1 in Canada and the United States among American countries (*16*), and from 1.3 in Greece and Hungary to 2.1 in Finland, France, and Belgium among European countries (*17*).

Further insight into the causes of the sex difference in coronary heart disease is provided by the striking contrast between the patterns of sex differences for coronary heart disease and for two other major cardiovascular diseases, namely, hypertension and vascular lesions of the central nervous system or stroke. Sex mortality ratios are much lower for hypertensive heart disease and stroke than for coronary heart disease—1.0 and 1.2 compared to 2.0 in the United States (*1*). Morbidity data show the same pattern of smaller sex differences for stroke and hypertension than for coronary heart disease (figure III–3). In international data, there is a similar contrast between the sex mortality ratios cited above for coronary heart disease and the sex mortality ratios for hypertension, which range from a low of 0.5 in Finland to a high of only 1.5 in Ceylon. The sex mortality ratio for stroke ranges from 0.9 in a dozen different countries to 1.4 in Ceylon and Japan (*16, 17*). Another contrast is that the sex mortality ratios for hypertension and for stroke are not correlated with economic development, while the sex mortality ratios for arteriosclerotic heart disease are (*16–19*). Death rates for stroke have declined for both males and females in the United States since the beginning of the 20th century with only a slight increase in sex mortality ratios, in marked contrast to the rising male death rates and rising sex mortality ratios for coronary heart disease (*20*). All these observations suggest that the factors primarily responsible for the sex differential in coronary heart disease must be factors which have little impact on hypertension and stroke.

In this connection, it is of special interest that the hard-driving coronary-prone behavior pattern increases men's risk of coronary heart disease, but does not significantly increase their risk of hypertension or stroke (*21;* S. Zyzanski, personal communication). In addition, cigarette smoking by men results in a greater increase in their risk of coronary heart disease than of hypertension or stroke (*22*). Both the coronary-prone behavior pattern (*23*) and cigarette smoking (*24*) have been linked to increased atherosclerosis of the coronary arteries. As would be expected, men in the United States have substantially more severe coronary atherosclerosis than do women (figure III–3). Sex differences in coronary atherosclerosis are much smaller in some other societies (*25, 26*), as would be expected if behavioral factors such as the coronary-prone behavior pattern and smoking are the primary causes of this sex difference. Thus, these new data corroborate previously presented evidence (*1*) for the importance of men's higher levels of coronary-prone behavior pattern and smoking as causes of the sex difference in coronary heart disease.

Detailed data from one large sample in the United States allow a more precise characterization of the contribution of cigarette smoking to the sex difference in coronary heart disease mortality (*22*). Depending on the age group, approximately one-quarter to three-fifths of the sex difference in coronary heart disease is related to cigarette smoking (table III–10). The contribution of cigarette smoking has several components: more men than women smoke cigarettes; male smokers smoke

Sex Differences in Cardiovascular Morbidity

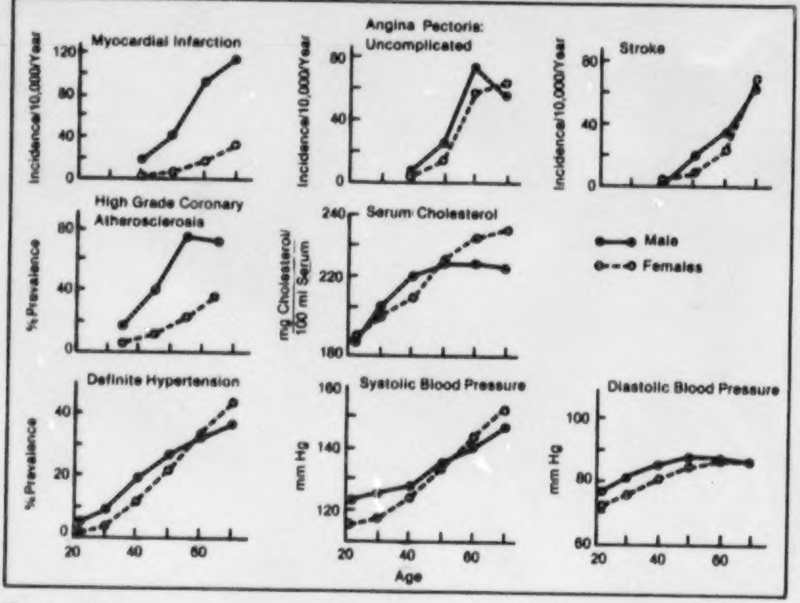


Figure III-3.
Sex differences in cardiovascular morbidity in the United States. Sex differences are particularly large for myocardial infarctions and for severe atherosclerosis of the coronary arteries. (The incidence data are from the Framingham Heart Study (*37*). Data on atherosclerosis are for a sample from Minnesota (*25*). Cholesterol and blood pressure data are for U.S. national samples (*39*, *70*)).

more cigarettes, more of each cigarette and cigarettes with higher tar content; men inhale more; and men may be more susceptible to a given dosage pattern of cigarette smoking (*22*, *27*, *28*). These effects are so confounded that it is impossible to make an accurate estimate of the independent contribution of each factor. However, an initial analysis of the data indicates that sex differences in the prevalence of cigarette smoking make a relatively large contribution at older ages and that other factors appear to be more important at younger ages (table III-10).

To summarize, female sex hormones appear to protect them to some extent against coronary heart disease. Evidence that both genetic and cultural factors contribute to the sex difference in coronary heart disease is provided by international data; men have higher death rates in almost all countries, but the size of the sex difference is highly variable. Two lines of evidence lend further support to the argument that the coronary-prone behavior pattern and cigarette smoking are major causes of the sex differential in coronary heart disease. Both of these behavioral causes would be expected to produce sex differences in

Table III–10.
Relationship of sex differences in coronary heart disease mortality to cigarette smoking

Sex differences in death rates for coronary heart disease [1]	Age 45–54	Age 55–64	Age 65–74
Total sex difference (male minus female death rates)	291	634	976
Sex difference unrelated to smoking (male minus female death rates for nonsmokers)	117	379	747
Sex difference related to smoking	174	255	229
Sex difference related to greater prevalence of cigarette smoking in men			
Minimum estimate: sex difference in prevalence x difference in death rates between female cigarette smokers and nonsmokers	11	49	117
Maximum estimate: sex difference in prevalence x difference in death rates between male cigarette smokers and nonsmokers	84	175	219

[1] Numbers shown are deaths per 100,000 person-years. Data are from (*22*). Although this sample was not a random sample of the U.S. population, the characteristics of the sample with regard to smoking behavior are close enough to those for a national random sample (*27*, *25*) to allow reasonable extrapolation.

atherosclerosis which vary cross-culturally and lead to a larger sex differential in coronary heart disease than in hypertension or cerebrovascular disease. Both expected patterns are observed.

Risk Factors and Cardiovascular Morbidity

In the United States, men have higher levels of serum cholesterol than women at ages 30–45 and lower levels above age 50 (figure III–3). The causes of the sex difference at younger ages probably include the effects of female sex hormones, which lower serum cholesterol (*1*, *2*, *9*) and the coronary-prone behavior pattern and possibly work pressures, both of which elevate serum cholesterol (*21*, *30*). It is unclear why women have higher levels of serum cholesterol than men at older ages.

For systolic blood pressure the typical pattern of sex differences in the United States and in other industrial societies is that men have higher pressures than women at younger ages and women have higher pressures than men at older ages (figure III–3). The age at which the sex differential reverses from a male excess to a female excess varies between age 35 and age 55 depending on the sample (*31*, *32*). Sex differences in diastolic blood pressure are typically smaller and less consistent (*29*, *31*).

In international data, there is a fairly consistent pattern of higher systolic blood pressure for men than for women at ages 20 to 25, a steeper gradient of systolic blood pressure with age for women, and higher systolic blood pressure for women than for men at ages 60 to 65 (*32, 33*). However, there is considerable variation in the age at which the blood pressure of women first exceeds that of men. As a result, in some societies women show higher blood pressure over most of the adult age span, whereas in other societies, blood pressure of men is higher at most adult ages (*32, 33*). The amount of elevation of blood pressure in older people varies enormously among societies (*33*). There are, for example, undisrupted, traditional societies in which blood pressures are low and do not rise with age (*32*). These data suggest that cultural factors strongly influence the overall age trends of blood pressure levels and also influence the magnitude and, at many ages, even the direction of sex differences in blood pressure. On the other hand, there is a relatively consistent tendency for blood pressures of men to be higher than those of women at young adult ages and for blood pressures of women to show a steeper gradient with age. These sex differences may have a biological basis, although the causes remain obscure. Available data suggest that menopause does *not* lead to an increase in hypertension (*13*).

Kannel (*34*) has presented evidence that the standard risk factors are not primarily responsible for sex differences in cardiovascular disease. Figure III-4 presents evidence on the same question for the specific category of coronary heart disease. At the younger ages the sex ratios for men and women who have identical risk factors are much lower than the sex ratios for men and women who have the average risk factors for their respective sexes. This indicates that at ages under 50, sex differences in the standard risk factors do contribute substantially to the sex difference observed in coronary heart disease. At these younger ages, men's higher levels of cigarette smoking, serum cholesterol and blood pressure probably all contribute to their higher risk as compared to women. At older ages, women have higher serum cholesterol levels and tend to have higher blood pressures than do men. As might be expected, then, the sex ratio for coronary heart disease incidence and mortality is considerably lower at these older ages. At these ages, the net contribution of the standard risk factors to the sex difference appears to be minor (figure III-4), presumably because men's disadvantage due to their higher levels of smoking is offset by their advantage in serum cholesterol and possibly blood pressure.

Thus, the causes of the sex difference in coronary heart disease appear to include men's higher levels of cigarette smoking, factors which contribute to men's higher blood pressure and serum cholesterol levels at younger ages, and other factors which do not have their effect via the standard risk factors. This latter group of causal factors probably includes female sex hormones and the coronary-prone behavior pattern, both of which appear to act primarily via physiological changes not measured by the standard risk factors (*4, 21, 35*).

As can also be seen from figure III-3, sex differences are much larger for the incidence of myocardial infarctions than for angina pec-

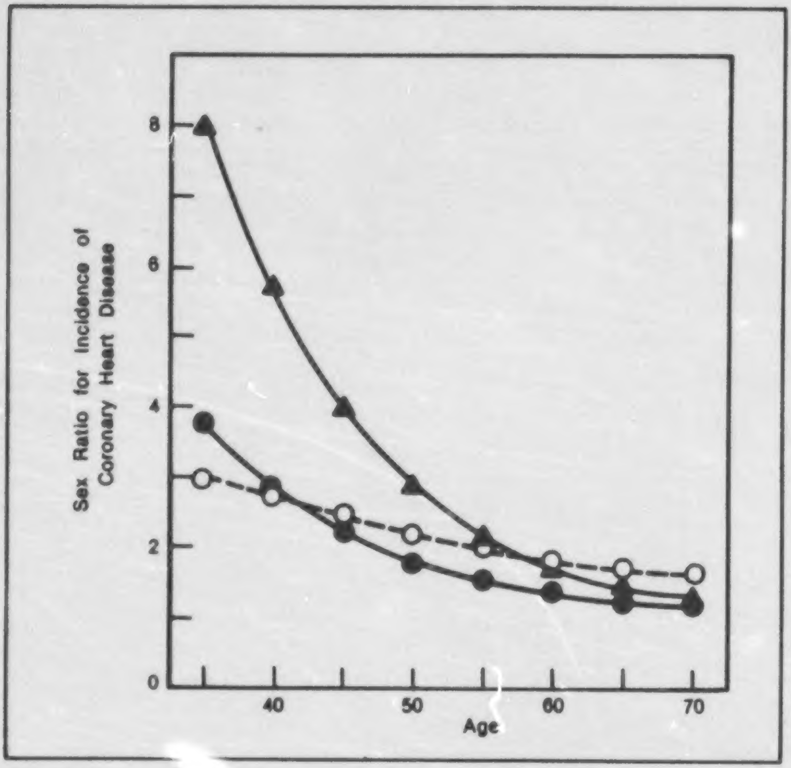


Figure III-4.

Sex ratios for the incidence of coronary heart disease for men and women with identical high (-●-) or low (-○-) levels of standard risk factors and sex ratios for men and women with the average levels of risk factors for their respective sex (-▲-). For the high risk group, systolic blood pressure is 195 mm Hg, serum cholesterol is 335 mg/100 ml, and cigarette smoking, glucose intolerance and left ventricular hypertrophy by electrocardiogram are all present. For the low risk group, systolic blood pressure is 105 mm Hg, serum cholesterol is 185 mg/100 ml, and smoking, glucose intolerance and left ventricular hypertrophy by electrocardiogram are all absent (data from *71*).

toris. Angina is more commonly the presenting complaint in women but more commonly a sequel of myocardial infarction in men (*36*). Cases of angina pectoris identified in women appear to be less severe than those identified in men (*20*, *36*, *37*). These findings may reflect a greater willingness of women to recognize and report the symptoms which lead to a diagnosis of angina (*31*).

An analogous result has been found in studies of hypertension. In health interviews, more women than men report that they have hypertension at all ages (*38*). In contrast, when hypertension is evaluated clinically (as systolic blood pressure of at least 160 mm Hg or diastolic blood pressure of at least 95 mm Hg), then hypertension is more prevalent in men up to age 54 (figure III-3). This pattern probably reflects not only greater sensitivity of women to symptoms, but also their more frequent visits to doctors (*2*), resulting in a substantially lower proportion of women than men who were unaware of their hypertension prior to a population survey (*39*). Studies of other types of morbidity have also shown that, for a given level of clinically established morbidity, women generally report more morbidity in a health interview than do men (*2, 38, 40*). Women also use preventive health services more and also reduce their activities more often when they feel sick (*2, 38, 40*).

It would be important to test whether the sensitivity of women to symptoms and their greater use of preventive services and rest contribute to their greater longevity. If so, can men learn these patterns, and can they be provided enough job flexibility so they can respond to symptoms and rest when ill?

Cross-Cultural and Historical Variation

The pattern of sex differences in mortality in the English-speaking industrial countries is similar to the pattern already described for the United States (table III-11). Males have excess mortality for coronary heart disease, ulcers, suicide, cirrhosis of the liver, accidents, cancer and tuberculosis. Again, this pattern suggests that men have higher mortality primarily because of a variety of behavior patterns harmful to their health.

The Scandinavian countries show a similar pattern, but with a smaller sex differential primarily because the sex differences in coronary heart disease are smaller (table III-11). This may be due to lower levels of cigarette smoking among men in these countries (*41, 42*). Several analyses have established that, among economically developed countries, those where men have high levels of smoking have larger sex differentials in mortality (*41, 42*).

The continental European countries, on the other hand, show a high sex differential but with a somewhat different pattern of causes, i.e., larger contributions from cirrhosis, suicide, accidents and tuberculosis. Ledermann (*43*) has shown that the high sex differentials for these causes of death are related to the high levels of alcohol consumption by men in the continental European countries.

Cross-cultural and historical comparisons between industrialized and nonindustrialized countries have yielded several consistent findings. Sex differences in longevity are smaller in predominantly rural, agricultural countries (table III-11) (*44-46*). Indeed, in many countries in the past, female mortality has exceeded male mortality between the ages of 1 and 40, both for total mortality and for a wide variety of specific causes of death (*45, 47*). The excess of male over female mortality increases during the course of economic development (*44, 47*).

Table III–11.
Causes of excess male mortality in different regions [1]

	Arterio-sclerotic and coronary heart disease	Ulcers	Suicide	Cirrhosis of the liver	Acci-dents	Tuber-culosis	Cancer	Total
English-speaking (Australia, New Zealand, Canada, United States, United Kingdom)	+309	+16	+17	+11	+51	+22	+28	+542
Scandinavian (Denmark, Netherlands, Norway, Sweden, Israel)	+156	+12	+19	+3	+38	+13	−10	+265
Continental European (Austria, Belgium, France, Germany, Italy, Portugal, Hungary, Switzerland)	+98	+17	+27	+30	+63	+62	+54	+515
Nonindustrial (Colombia, Ceylon, Honduras, Panama, Yugoslavia)	+19	+5	+24	+3	+54	+24	−31	+184

[1] Numbers shown are male minus female death rates (deaths per 100,000 persons per year) for persons aged 45–64, 1954–1955. (Adapted from reference *43*.)

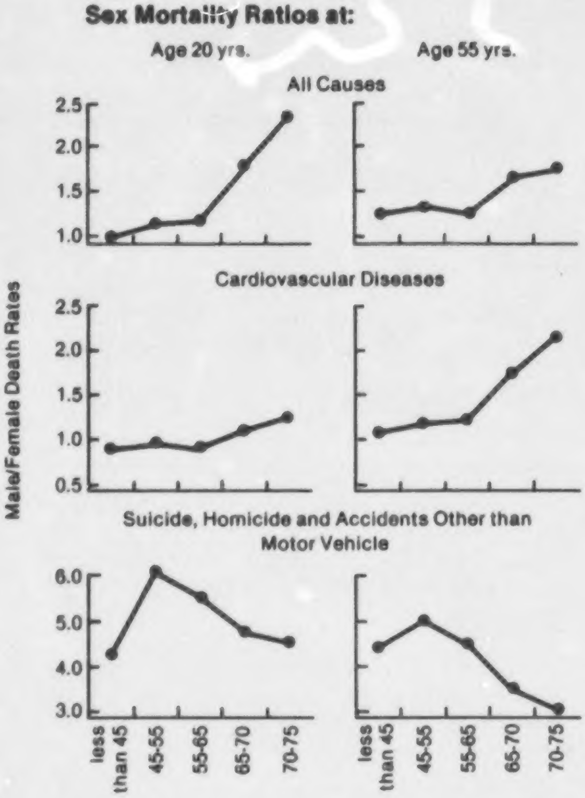
The rise in sex mortality ratios is particularly marked for young adults, aged 15 to 24 and for older adults, aged 45 to 64 (*44, 48*). At older ages, the largest contributors to the rise in sex mortality ratios during industrialization are the rise in the male excess of cardiovascular diseases and cancer (*48–50*). These trends are reflected in the rise of sex mortality ratios with the increase in life expectancy which is illustrated in figure III–5.

The increases in sex mortality ratios for cardiovascular diseases and cancer are due primarily to increases in the rates for men of coronary heart disease and lung cancer with industrialization (*25, 48, 51, 52*). One cause of these trends is an increase in cigarette smoking, especially for men (*49, 50*). Other factors which have contributed to the increase of sex differentials in mortality with economic development are improved medical care resulting in decreased maternal mortality and possibly the effects of decreased discrimination against women and decreased exercise, increased exposure to industrial carcinogens, and increased prevalence of the coronary prone-behavior pattern for men (*1, 2, 48–50*).

Other mortality trends during the early stages of industrialization provide further insight into the conditions experienced by men and women during this period. Sex mortality ratios for suicide, homicide and accidents other than from motor vehicles increase from the lowest level of life expectancy to the next level (figure III–5). This may be due to an increase in factory accidents for males during early industrialization or to a rise in suicide disproportionately for males. For example, in Sweden, during the 19th-century period of early industrialization and urbanization, male death rates for suicide rose from 3.4 to 25.4 per 100,000 population per year and the sex mortality ratio for suicide increased from 3.0 to 4.7. (This increase occurred between 1785 and 1905 as life expectancy rose from 36 to 56 years. For this very early period, it was not possible to calculate age-adjusted death rates (*53*)). After 1905, sex mortality ratios for suicide began to fall, while sex mortality ratios for ulcers rose due to a substantial increase in the rates for ulcers among men (*54*).

The trends during the early 20th century were similar in the United States, where sex mortality ratios for suicide fell after 1900 while ulcers showed an increasing male predominance (*54, 56*). During this period, sex mortality ratios for cirrhosis of the liver and homicide also rose (*55*). Other international comparisons provide suggestive evidence of similar trends. For example, in a sample of North and South American countries, the sex mortality ratio for cirrhosis of the liver increases with a rise in the per capita GNP (*16, 19*).

A definite interpretation of these trends will depend on more detailed historical analyses. However, the trends are suggestive of rising stress experienced by males during the very early period of industrialization, as indicated by the early rise in suicide, with a subsequent shift of male stress-related mortality to ulcers and alcohol-related causes, such as cirrhosis.



Life Expectancy

Representative countries in each life expectancy samples are:

less than 45 - Chile, 1909-1940; England and Wales, 1861-8171; Taiwan, 1920-1936

45-55 - Chile, 1950-1959; England and Wales, 1901-1911; U.S., 1900-1910

55-65 - England and Wales, 1921-1940; Taiwan, 1960; U.S., 1930-1940

65-70 - England and Wales, 1951; Taiwan, 1964; U.S., 1960

70-75 - England and Wales, 1960-64; Sweden, 1951-64; U.S., 1964

Figure III–5.
Sex mortality ratios at different levels of life expectancy. Sex mortality ratios for all causes and for cardiovascular diseases are higher in countries where mortality is lower. Sex mortality ratios for suicide, homicide and accidents other than motor vehicle peak for life expectancies between 45 and 55 (*45*).

Thus, among industrial countries, those with high cigarette smoking or high alcohol consumption have larger sex differences in mortality. During industrialization, the sex differential in mortality has shown increases. These increases appear to be related to urbanization, increased smoking by men, improved health care which has benefited women particularly, and possibly increased stress for men and decreased discrimination against women.

Variation in the United States

Sex mortality ratios for middle-aged whites are particularly high in the South (*57*). In this region, these ratios are high for cardiovascular-renal diseases, malignant neoplasms and total death rates (*58*). One possible cause of the higher sex differentials in the South may be stress experienced by men in connection with high levels of violence. Homicide rates are double those in other regions of the United States (*59*) and gun ownership is 50 percent more common (*60*). Figure III–6 presents a preliminary test of the hypothesis that a culture of violence produces stress that contributes to excess male mortality. This figure shows that high sex mortality ratios for middle-aged whites occur in States with high homicide rates. This relationship is not due to the direct contribution of homicide to the death rate, since homicide is a minor cause of death in this age range. Many other factors may contribute to the increased sex mortality ratios in the South and a more systematic analysis of this phenomenon would be of interest.

The sex differential in life expectancy by race has been lower for blacks than for whites during much of the 20th century. However, during the 1960's the life expectancy of black females rose rapidly while the life expectancy of black males remained stationary, so the sex differential in life expectancy increased rapidly and now is at least as great as the differential for whites (*61*). The life expectancy of black males failed to rise during the 1960's primarily because death rates for adult black males *increased*, due to an increase in the death rates for arteriosclerotic heart disease, vascular lesions of the central nervous system, malignant neoplasms of the respiratory system, emphysema, cirrhosis of the liver, suicide, homicide and accidents (*62*). These trends suggest underlying social trends which adversely affected black males during the 1960's.

Although the sex differential in life expectancy is now similar for whites and blacks, the components of the differential are different for the two races. Whites have higher sex mortality ratios for heart disease and for bronchitis, emphysema and asthma; in addition, the proportion of total deaths due to these causes (which have high sex mortality ratios) is higher for whites than for blacks (*63*). In contrast, blacks have higher sex mortality ratios for motor vehicle accidents and a higher proportion of deaths due to homicide and several other causes which have high sex mortality ratios. This difference in mortality patterns resembles the difference described previously between the English-speaking and continental European countries.

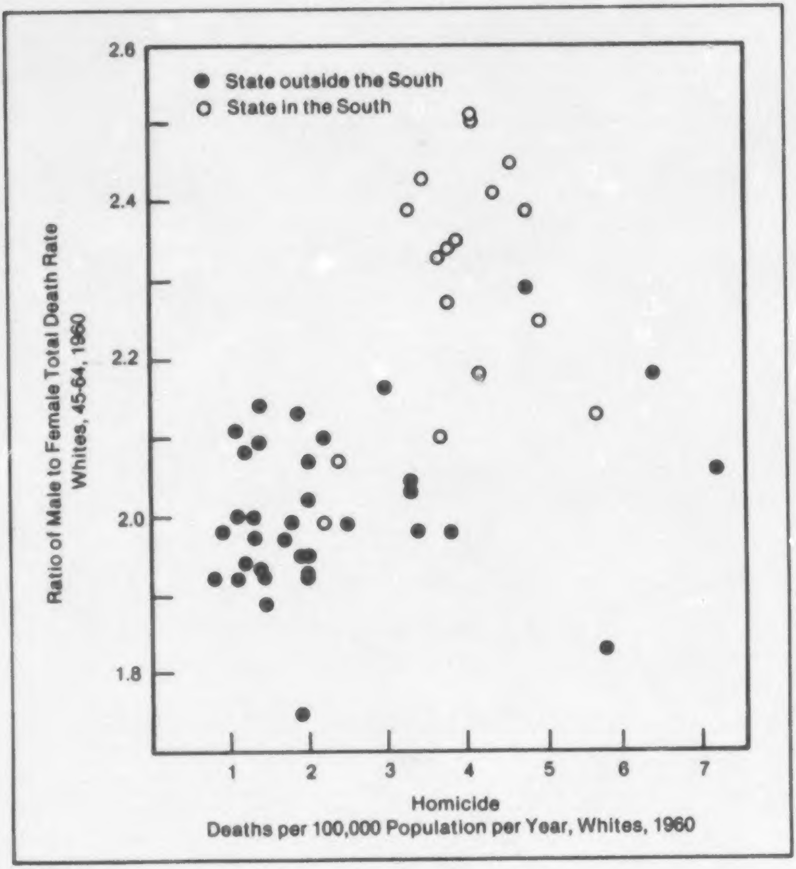


Figure III–6.
Sex mortality ratios compared to homicide rates in the United States. States with high homicide rates have higher sex mortality ratios (*57*, *59*).

On the basis of the analyses presented in this paper, one would expect the life expectancy of women and men to begin to converge, since, in the last half century, growing numbers of women have begun to smoke cigarettes, drink alcohol and enter the labor force (*2*). Such a trend is not yet evident, but there are hints that a trend of decreasing sex differences in mortality may be developing. Sex mortality ratios for malignant neoplasms of the respiratory system and suicide have decreased recently as female death rates for these causes have increased (*38*, *55*, *56*). Sex mortality ratios for ulcers have also decreased (*38*).

Are these trends early harbingers of a more general deterioration of women's health? Future mortality data will provide the answer to this important question.

Future Research

Specific questions and hypothesis for testing have been presented in previous sections of this paper. Several general questions and approaches should also be pursued through future research. Men have a higher mortality rate than women in industrial societies because of a wide variety of behaviors including cigarette smoking, alcohol consumption, driving habits, use of guns, exposure to job hazards and the hard-driving style of life known as the coronary-prone behavior pattern. Why are so many different aspects of men's behavior detrimental to their health? Inherent sex differences in aggressiveness may contribute to the behavioral differences described, but cross-cultural studies indicate that this contribution is probably less important than the contribution of cultural factors (*1, 2*). This raises the question: What feature of industrial society results in such a wide array of unhealthy behaviors occurring predominantly in men? One hypothesis is that these diverse behaviors are all increased by pressures related to employment or by childrearing methods which serve to adapt males to the role demands of bread winner in industrial society (*1–3*). This hypothesis should be tested.

The effects of employment on health and health related behavior are more easily assessed for women than for men, since housewives form a useful comparison group. One approach might be an analysis of the relationship between the labor force participation rate of the women in the labor force and the condition of their health in different regions. Another approach has been to compare the health and behavior of employed women and housewives. The results of such studies have been interesting, but somewhat ambiguous. Employed women show more of the hard-driving coronary-prone behavior pattern than housewives do, but it is not clear whether this is because hard-driving women seek employment or because of the effects of employment on women (*3, 64*). Employed women are generally found to be healthier than housewives (*64, 65*), but this may simply reflect the fact that less healthy women are less likely to obtain or keep paid jobs (*64*). Such an effect has been observed in men (*66*).

Longitudinal studies of women during a period when they begin or end paid employment could provide more information on the causal relation between work and health and related behavior patterns. In such studies it would be important to obtain information about each woman's individual situation—for example, concerning her attitude toward work, the satisfactions and pressures she experiences at work, conflicting time demands and social support. These factors probably influence the effect that employment has on health. Suggestive of such effects is the finding that among long-term telephone operators, the unhealthiest were those who were frustrated because they had been unable to marry and leave the job, whereas the healthiest had not aspired to marry and liked the work because it was not too demanding (*67*).

Since cigarette smoking and alcohol consumption are frequently implicated as factors responsible for male excess mortality, it would be useful to explore the reasons why, in most populations, more men than women smoke cigarettes and drink alcohol excessively. Social pressures appear to play a role, and sex differences may disappear when not reinforced by social mores. For example, cigarette smoking is approximately the same for teenage girls and boys at the present time in the U.S. (*68*). Similarly, alcohol consumption was equal for the two sexes in one-third of a large sample of nonindustrial societies (*69*). This raises the question: Why have cigarette smoking and alcohol consumption been more acceptable for men than for women in so many societies? One line of approach is suggested by the following hypothesis: Bacon (*69*) notes that equal alcohol consumption for women and men has been particularly common in societies where alcohol has been introduced recently. She hypothesizes that social proscriptions against heavy drinking by women develop in time because inebriation can interfere so seriously with women's child-care role. Other factors are probably also important and should be explored. For example, both smoking and drinking appear to be linked to characteristics which are more often associated with the male role, e.g., rebelliousness and pressures to be independent and to achieve (*1*, *2*, *68*).

Another interesting question for further research is an evaluation of the relative importance of the factors which may contribute to the increase in the sex mortality ratio with industrialization. One useful approach would be to examine the relationship between sex mortality ratios for specific causes of death and a variety of socioeconomic indicators over a wide range of historical conditions. Arteriosclerotic heart disease has been a major contributor to the trends described and would be a particularly interesting candidate for this type of analysis. In addition, systematic analysis of detailed causes of death such as suicide and cirrhosis of the liver might provide interesting insights into the conditions experienced by men and women at different stages of industrialization. Specifically, this type of analysis would provide a useful approach to the question of whether men experience increased stress during early industrializaton. It might prove fruitful to analyze the relations between mortality and specific working conditions. For instance, death rates for men employed in specific industries could be examined to see whether rises in death rates occur during periods when accelerated work pace is introduced in that industry.

Summary

In industrial countries, men have higher mortality rates than women, particularly for causes of death with a major behavioral component. In the United States, a third of the sex difference in mortality is due to

higher rates for men of respiratory cancers, emphysema, motor vehicle and other accidents, suicide and cirrhosis of the liver. Forty percent of the sex difference is due to coronary heart disease. These sex differences in mortality are to a large extent the consequence of behaviors which are more common in men, including cigarette smoking, alcohol consumption, use of guns, exposure to job hazards, and the hard-driving coronary-prone behavior pattern.

Sex differences in coronary heart disease mortality and coronary atherosclerosis vary cross-culturally and are much smaller in some other countries than in the United States. This indicates that cultural factors have a major influence on sex differences in coronary heart disease. Two types of culturally influenced behavior, cigarette smoking and the coronary-prone behavior pattern, appear to play a particularly important role. Higher levels of cigarette smoking and the coronary-prone behavior pattern among men would be expected to result in substantial sex differences in coronary heart disease and coronary atherosclerosis, and small sex differences in stroke and hypertension. This is the pattern of sex differences in cardiovascular disease which is observed.

A protective effect of female sex hormones may also contribute to the sex difference in coronary heart disease. Both the sex hormones and the coronary-prone behavior pattern appear to act in part via their effects on serum cholesterol, but to a greater extent via physiological changes not reflected in the standard risk factors.

Sex differences in longevity are smaller in non-industrial societies than in industrial societies. To a large extent, this reflects lower sex mortality ratios for coronary heart disease in the non-industrial societies. During the early phase of industrialization, sex mortality ratios for stress-related causes of death such as suicide, ulcers and cirrhosis of the liver appear to rise. These trends during industrialization may be due to increased cigarette smoking particularly among men, improved status for women, improved medical care which lowered women's mortality particularly, and increased stress for men during early industrialization.

The trend toward increasing sex differentials in longevity may be reversed in the United States in the future. Sex mortality ratios for ulcers, suicide and cancers of the respiratory system have all declined since 1960 as women's death rates for suicide and respiratory cancers have risen.

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Discussion

Costa: I would like to take issue with your interpretation that sex difference in smoking is attributed to personality. I believe you said that increased smoking among men is due to greater incidence of either rebelliousness, aggressiveness, or impulsiveness. Two factors need attention. The first is that more than a score of studies have not been able to delineate a smoker personality.

The second factor is that there has been a tremendous increase in the rate of cigarette smoking among women since the 1900's. We would interpret that fact, according to your logic, by saying women are becoming more rebellious when a more parsimonious interpretation might be that there are cohort differences, sex role changes, and proscriptions about smoking.

Waldron: I have two comments. One is that I think the cohort phenomenon is important and I do describe that in my more detailed analysis and did not have time to include it today. I think that you are quite right; sex differences are largest for older people and this is probably due to the fact that smoking was culturally unacceptable for women at the time that these older women were teenagers, which is the age when a person is most likely to start smoking.

A very interesting study on young women, just published by the American Cancer Society, shows again that personal characteristics may be important. Various indices of rebellious behavior, such as getting expelled from school are much higher in young women who smoke than in young women who do not smoke.

Costa: But that could be a sign of maladjustment or the failure to cope and not a personality trait.

Waldron: I am not talking about personality traits, I am talking about personal characteristics. With regard to the relationship between cigarette smoking and cancer, I do not want to get involved in a big argument about whether we can show that cigarette smoking causes lung cancer. In analyzing the importance of sex differences in smoking habits, there are a couple of things to consider. First, more data are needed to determine whether a given level of smoking causes the same risk of lung cancer in men and women. Second, currently available data indicate that the difference in the cigarette smoking levels between men and women can account for a substantial proportion of the sex difference in lung cancer and coronary heart disease death rates.

Palmore: What would be your estimate, if you care to make one, of the proportion of the sex differential that you can account for by sex-linked genes versus behavioral life-styles?

Waldron: I do not believe the data are available to make anything more than a ball park estimate. I would estimate that three-quarters of the sex difference in this country is due to cultural factors. However, I do not think the data are there to defend that estimate.

Palmore: One further point of clarification on your figure III–5 regarding the sex mortality ratio at ages 20 and 55, I do not understand what is along the base. It looks like you have age along the base.

Waldron: No, that is life expectancy. The reason it is graphed in this way is that the data are presented in that form in the source. If you look below, you can see that the sample countries illustrate the economic level which is typical for each life expectancy.

Gruenberg: While we are on that figure, could you explain the upper left hand corner? Does that first dot really suggest that at age 20 in the lowest life expectancy countries, women die faster than men?

Waldron: No, the risk is the same for men and women, with a ratio of one.

Siegel: You have been dealing mainly with specific causes. There are some more general phenomena operating that tend to lean on the side of greater weights for the role of biological factors. Among these are the excess fetal and infant mortality of boys and the fact that since the beginning of this century, there has been a tremendous shift in the causal pattern of mortality, which has in effect reduced sharply the role of infective and parasitic illnesses. As a result, the causal pattern is now dominated by the chronic or endogenous kinds of illnesses which, by their nature, are more likely to have a genetic basis.

There is also the view that one of the main factors in the change in the sex differential over time has been the increase in the length of the female reproductive period. As age at menarche has lowered and age at menopause has risen (a difference reflected in the difference in the length of the female reproductive period for the developing and developed countries), you also see a shift in the relative balance of male and female mortality. Evidently the reproductive period is a highly protective period for women—a period which grants women a greater resistance to cardiovascular illness in particular. Could it be that there is a social factor involved in that this is a period in which women also begin to see doctors regularly for prenatal care? After the reproductive period, rates of cardiovascular disease increase sharply for women.

Cohen: I think you are speaking from evidence that just is not there. I think if you look at the WHO data on sex differentials, particularly on cardiovascular disease mortality but also on total mortality, the peak male-female differential varies among countries anywhere from the twenties to the sixties and seventies.

If you are claiming that the reproductive period is a period of maximum protection, then you have got to have some very funny notions about differences in age of menopause in different countries that exceed what you are saying is the difference between developing and developed countries. The cross cultural data simply do not support the reproductive period hypothesis.

Kannel: The female immunity to cardiovascular diseases, I am persuaded, cannot be explained by any differences in the major identified risk factors. At any level of smoking, women are better off than men for occlusive peripheral arterial disease, stroke or congestive heart failure. This is true for everything except diabetes, which somehow seems to eliminate the female immunity to cardiovascular mortality.

The other thing which I think is quite clear is that the gap in incidence between the sexes narrows progressively with advancing age, suggesting that something is happening to women which is unfavorable as they get older.

Cohen: Or, alternatively, something favorable is happening to men as they get older. In fact, the curve for men rises rapidly and levels off and the one for women goes up steadily with increasing age.

Kannel: Yes, that is a possibility except for one thing. We have some data which indicate that women who undergo the menopause almost triple their cardiovascular risk compared to women of the same age who stay premenopausal.

Speaker: I am not familiar with the details, but I understand that there are data that show generally better life expectancy among Mormons and Seventh Day Adventists who are very strict about not smoking and drinking. Is this reflected in a drop in the male-female mortality ratio?

Waldron: To the extent that I am familiar with the data, yes.

Feinleib: Dr. Waldron, you and several others predicted that there would be a convergence for the male-female mortality rate. But, during the last 30 years, the gap in overall life expectancy has widened; in fact, it seems to be widening at an accelerating rate during the last 6 or 8 years. When do you expect the convergence to show up and at what age ranges should we be looking for it?

Waldron: I cannot be very precise about my predictions. All I can say is that you do see the convergence in lung cancer, suicide and ulcers. Since about 1958, there has been a convergence in death rates for those three causes. Suicide and ulcers particularly may be causes which respond to changing social conditions rapidly and lung cancer may be the one that reflects very clearly the smoking trend. So these data suggest that we might see some decrease in sex differentials in the next couple of decades.

Siegel: None of them is a major cause of death.

Waldron: Right. So the overall mortality differential continues to increase.

Gibson: It seems likely that the difference we have is even more basic than that. In almost every animal species studied, the female usually lives longer.

Waldron: That is a common fallacy.

Gibson: Not according to the colonies we maintain.

Waldron: I have with me the original paper in which I concentrated on several ecological studies in natural environments, which is slightly different from what you were saying. There are as many cases where the male outlives the female as vice versa. I think there has been a tendency to focus on the few species in which males have higher mortality in laboratory studies. In field studies, however, the reverse is just as common.

Gibson: But you have a different massiveness, a different size of animal. I do not want to go too far afield on that, but predators and other factors play a role in the wild as opposed to a controlled environment where essentially all animals are equal. Under those conditions, in almost every species of animal, the female outlives the male.

Sacher: I would like to state my agreement with Dr. Waldon because I do not think the evidence is quite that consistent with regard to female superiority. There are a lot of species, and also some strains of mice and rats, which show no sex differential or a slight male advantage.

Survival After Early and Normal Retirement*

Suzanne G. Haynes, Ph.D.

Epidemiology Branch
National Heart, Lung, and Blood Institute
National Institutes of Health
Bethesda, MD 20205

Anthony J. McMichael, M.D., Ph. D.

Division of Human Nutrition
CSIRO
Adelaide, Australia 5000

H. A. Tyroler, M.D.

Department of Epidemiology and
Occupational Health Studies Group
University of North Carolina
School of Public Health
Chapel Hill, NC 27514

In 1935 the U.S. Congress passed the Social Security Act which institutionalized retirement by establishing the minimum age for retirement benefits at 65 years (*1*). Although this decision was somewhat arbitrary (*2*), the proportion of all work terminations officially regarded as "normal" retirements at age 65 has increased considerably since that time. Of all Americans terminating their working lives during the past 25 years, the proportion required to retire at a fixed age has tripled from 11 percent in 1951 (*3*) to 36 percent in 1975 (*4*). "Early" retirements (nonhealth related) before age 65 have also become more prevalent. In several industries, such early retirements have now replaced compulsory retirements as the most prevalent form of permanent separation from the work force (*5, 6*).

Unfortunately, the rationale and health consequences of the 1935 Social Security Act have not been sufficiently explored. In 1940 the average expectation of life for a male surviving past the first year of life was 65 years (*7*), whereas today the comparable expectation is 68 years (*8*). Increases in life expectancy have occured since 1900, so that a man surviving to age 65 can, on the average, expect to live 13.1 additional years (*9*). The trends suggest that persons aged 65 will be surviving even longer in the future.

The effect on subsequent survival of terminating one's work productivity at age 65 has not been thoroughly explored. Several investigators, including the AMA Committee on Aging, have suggested that normal retirement may initiate or accentuate medical problems (*10, 11, 12*). Evidence to substantiate such claims, however, has not been found in past studies (*13, 14, 15*), although methodological weaknesses were apparent in most of these studies (*16*). Likewise, factors related to survival after early and normal retirement have not been explored. We might expect that predictors of mortality after normal retirement would be different from those after early retirement, especially if the reasons for each retirement are different. Likewise, predictors of mortality in a general population may be different from predictors in a working population. Selection of persons into the latter group implies that they are more healthy than the general population (*17*).

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In the study described below, mortality experiences of a large cohort of blue-collar workers in the rubber tire industry taking normal (age 65) and early retirements (age 62 to 64) are compared. In this particular industry, retirement may take place for any one of three reasons: work-related medical disabilities (disability retirement); individual preference to retire before the mandatory age limit (early, voluntary retirement); or a fixed-age limit for work in the industry (normal, involuntary retirement). Theoretically, the problems and life crises associated with the process of retirement might be expected to apply to all types of retirements. However, recent studies in a variety of settings, and with a number of measures, have shown early retirees to be significantly more satisfied with retirement than normal pensioners (*18, 19*). Factors related to mortality after the two latter forms of retirement are outlined and compared to correlates of survival in other populations. In general, this study describes the patterns and correlates of survival after normal and early retirement in the hopes of assessing the effects of our current national policy of retirement at or before age 65.

Hypotheses

Based on previous research in this area, three hypotheses were tested.

Hypothesis I

> If retirement poses stressful experiences for workers forced to retire at age 65, mortality will be higher than expected at some stage during the first 5 years after retirement.

Although no actual measures of "stress" were available in this study to test hypothesis I, Atchley (*20*) has identified several phases in the retirement process which are helpful in conceptualizing a stress hypothesis. The first phase after the retirement event is thought to be the "honeymoon phase," a euphoric period in which the individual revels in his newly acquired freedom of time and space. The honeymoon phase is thought to be followed by a "disenchantment phase," a period of dissatisfaction and adjustment problems. The disenchantment period is most likely caused by inadequate pensions, loss of friends, and loss of an occupation.

The disenchantment phase has most relevance for a stress hypothesis since individuals may suffer from depression, nihilism, or resentment during this period (*21*). The exact timing of the honeymoon and disenchantment phases, if they do exist, has not been documented in longitudinal studies. However, they are most likely to occur at some stage during the first 5 years after retirement. For this reason, we will examine death rates during specific intervals of time (yearly) after retirement. Since it is possible for mortality rates to be quite low during the hypothesized honeymoon phase, and excessive during the so-called disenchantment phase, an average 5-year mortality rate after retirement would disguise any temporary increase or decrease in mortality during these phases.

Finally, if one seeks to study the health effects of psychosocial stress associated with retirement—involuntary, nonmedical retirements appear to afford the best research opportunity.

Hypothesis II

> If early retirements before age 65 are taken by some workers because of failing health, mortality will be higher than expected at some stage during the first 5 years after retirement.

There is little question that health plays an important role in the early retirement decision for some persons. Barfield and Morgan (*5*) reported that 24 percent of automobile workers planning to retire before age 65 gave health as a reason for retirement. If health is important in the decision to retire early, it is also likely to be an important predictor of death immediately following retirement and to be reflected in higher than expected mortality rates in this period.

Hypothesis III

Those persons more likely to die within 5 years after a normal or early retirement will:

(a) have lower social status before leaving the work force,
(b) have poorer health status during their working career, particularly within the 2 years before retirement,
(c) experience more job dissatisfaction, and
(d) have less social support from family and others than do persons who survive over 5 years after the retirement event.

The relative importance in premature death of such factors as socioeconomic status, job satisfaction, and social supports comes from several studies in the longevity and mortality literature (*22*, *23*).

Methods

As previously described (*16*), the data presented here derive from the Akron, Ohio plants of two U.S. rubber tire companies, and refer exclusively to deaths among male, blue-collar workers, i.e., hourly (factory) employees.

Initially, a historical prospective design was used in the present study. The original cohort (or population at risk) comprised 2,129 individuals taking normal retirements at age 65, and 1,842 individuals choosing early retirements between ages 62 and 64. These cohorts derived from active workers, aged 56 to 64 on January 1, 1964, followed for 9 years through early or normal retirement. Methods used for cohort identification and complete death ascertainment have been described elsewhere (*24*, *25*). Although early retirement can occur between the ages of 55 and 64, only workers choosing an early retirement at ages 62 to 64 were studied. The older early retirees were chosen because full pension benefits may be received from age 62 onward, and because most early retirements have occurred after age 61.

In addition to the descriptive mortality analyses to test hypotheses I and II, separate case-control studies for normal and early retirees were subsequently carried out in company A to elucidate the patterns of post-retirement mortality, and to explicitly test the four parts of hypothesis III related to survival after retirement.

The 110 deaths within 5 years of normal retirement comprised the *first* set of cases. Two hundred and twenty controls were randomly selected from the remaining 558 normal retirees who survived for 5 years or more after retirement. The *second* set of cases comprised 52 early retirees who died within 5 years of retirement; their controls consisted of 104 age-matched retirees who survived early retirement by more than 5 years.

The primary technique used for assessing the significance of the observed mortality within each cohort was to compare it to mortality expected in a working population after retirement. Since we were concerned with increased death rates due to the process or circumstances of retirement, the ideal comparison population would be a group of blue-collar workers continuing employment after age 62 in the case of early retirement and after age 65 in the case of normal retirement. Published mortality rates for the latter group are not available for the United States. Using death rates from the U.S. population for comparison is also considered inappropriate because of the "healthy worker effect," wherein employed cohorts are noted to have better life expectancy than the general population, which includes nonemployed, hospitalized, and institutionalized persons (*17*).

For these reasons, mortality rates of actively employed male workers (under 65 years) and nondisabled retirees (over 65 years) were used as "expected" rates for our early and normal retiree studies, respectively. These rates were based on the pooled experience of workers from several large industrial firms (nongovernmental and nonbanking) in the United States. Since the "expected" rates for normal retirees do not represent the experiences of working populations over 65, their actual level may be higher than the "ideal" standard.

Data collected in this study were restricted to information available in company pension, personnel and medical records. As contact with individual pensioners or their surviving dependents was not possible, surrogate measures of some conceptualized variables were necessary.

Income, in the form of the last hourly pay rate, represented the basic rate of pay given an employee commensurate with the skill, responsibility, and hazards of the job. Occupation, as defined by the longest job held in the rubber industry, was categorized into highly skilled (skilled craftsmen, inspectors, and machine operators), skilled (tire builders, repairmen, and servicemen), semi-skilled (millmen, curemen, compounders, finishing men, and truckers), and unskilled (utility and service, machine and other helpers, and cleaning) groups. Social status was defined by the separate components of education, occupation, income, and local property ownership.

An index of morbidity in the 2 years before retirement consisted of the combined information on insured sickness absences (documented illnesses of over 7 days), hospitalizations, and health-related work limitations. A *low index of morbidity* was defined as no insured sickness absences and no hospitalizations and no work limitations during the 2 years before retirement. A *high index of morbidity* meant the presence of any or all of the three medical events mentioned above. Uninsured sickness absences were absences of less than 7 days duration, not requiring medical verification from a physician. The uninsured absences therefore represent absences from work due to a variety of reasons, including illness, job dissatisfaction, and unapproved vacation.

The primary statistic reported in this study is the relative odds, an approximation of relative risk. Relative risk refers to the ratio of the rate of disease (or death) among those "exposed" to a particular factor over the rate among those not so exposed. In a case-control study, this risk is estimated by the formula ad/bc from a standard 2 x 2 table (*26*). A risk is different from 1.00 suggests that an association of exposure and disease exists. In the early retirement case-control study, the Mantel-Haenszel (*27*) procedure was used to estimate the overall relative risk across the three-age strata. A computer program was used to calculate the exact confidence intervals around the relative risk estimates (*28*).

Findings

Figure III–7 shows the pattern of mortality after normal, involuntary retirement at age 65 (hypothesis I). Based on an extensive literature regarding retirement and stress (*16*), we postulated that mortality (while, admittedly, an inadequate measure of stress maladaptation) would be higher than expected at some stage during the first 5 years after normal retirement.

Figure III–7 shows that the age-specific mortality rates after retirement in each company exhibited considerable fluctuation around the expected rates. In company A, qx values were 18 percent greater than expected during the fourth year after retirement, although not significantly different. In company B, observed death rates were 30 percent higher than expected during the third year after retirement (significant at the $p=0.03$ level). When the two companies were pooled, to increase numbers, the observed rates were lower than expected in the first 2 years, about equal to expected for years 3 and 4, and subsequently

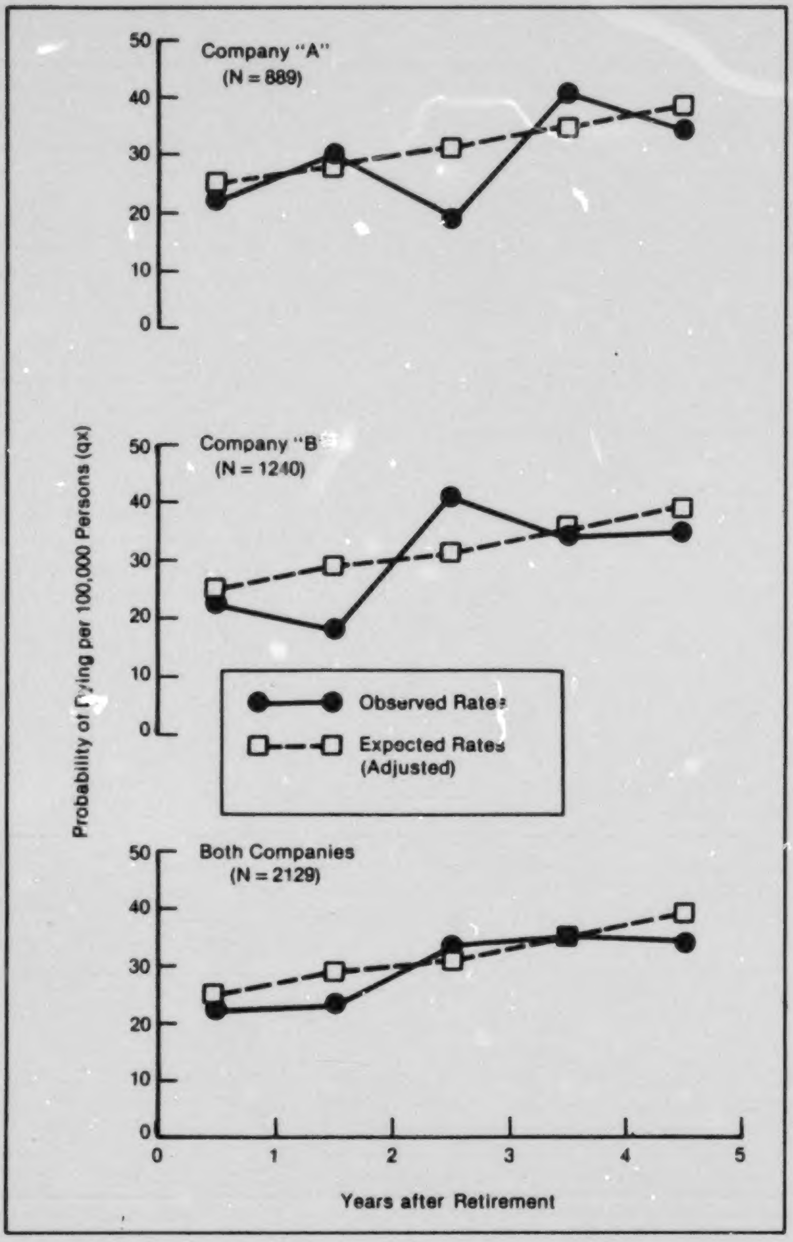

Figure III-7.
Mortality rates after normal retirement.

lower again. This trend is not inconsistent with the notion that years 3 and 4 may be the disenchantment years—especially since the expected rates were somewhat conservative, i.e., high, and the relevance of their absolute values is debatable.

Figure III–8 shows mortality rates among two cohorts of normal retirees from all the U.S. plants of company A. The first cohort includes persons who retired between the years 1953 and 1962. This cohort includes a mix of workers—those who wished to remain employed until age 65 and those who wished to retire early but were unable to do so for economic reasons, the benefits to early retirees being reduced during that time period. The second cohort contains workers retiring between 1963 and 1972. This group probably represents persons who desired a normal retirement at age 65 since full pension benefits were accorded to early retirees (between the ages of 62 and 64) during this period.

Examination of the mortality rates in the 1953 to 1962 cohort indicates a rather smooth rise in mortality after retirement. However, among persons retiring between 1963 and 1972, mortality rates were significantly lower than the 1953 to 1962 rates in the third year, and slightly higher than the 1953 to 1962 rates in the fourth year after retirement. These findings suggest that the disenchantment phase is

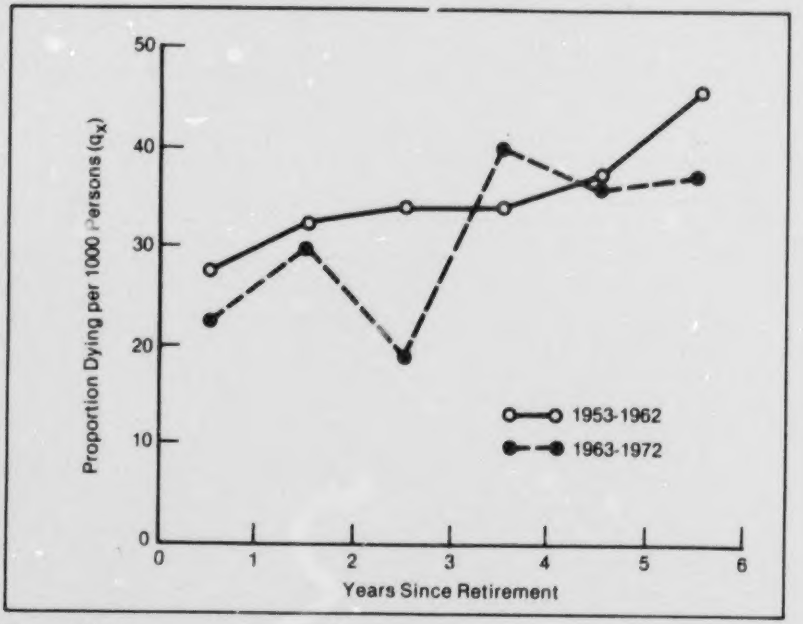

Figure III-8.
Mortality among normal retirees throughout all plants of a large rubber company, 1953–1962 and 1963–1972.

observable only among persons who desired to work until age 65, since earlier retirement was economically feasible during the 1963 to 1972 period.

Mortality rates after early retirement at ages 62 to 64 are shown in figure III–9 (hypothesis II). Here we postulated that mortality would be elevated after early retirement because of self-selection by persons in poor health into this type of retirement.

The pattern of mortality after early retirement follows a fairly similar U-shaped curve in both companies. Compared to the expected rates, the pooled death rates are significantly higher in years 1, 4, and 5. In the first year after retirement, the observed rates were two times greater than the expected rates. In years 4 and 5, the observed rates were approximately 40 percent greater than expected.

The relative risk estimates for survival according to five socioeconomic status variables among normal and early retirees are summarized in table III–12 (hypothesis III). In the first set of comparisons (normal retirees) cases occurring within 3 years of retirement were compared to controls (see group 1). Likewise, cases occurring 4 to 5 years after retirement were separately compared with the surviving controls (see group II). The two sets of comparisons were made to distinguish the predictors of mortality immediately following normal retirement from predictors during the later period when mortality rates were slightly elevated. Odds ratios were calculated with the lower status persons as the referent (or "nonexposed") group.

Low socioeconomic status was found to be predictive of subsequent early mortality only among normal retirees. As one might expect, lower

Table III–12.

Risk of early death after normal and early retirement by socioeconomic levels

Variables	Normal retirees		Early retirees [3]
	Group I [1]	Group II [2]	
1. Education (none vs. high school)	1. 86	1. 00	1. 33
2. Pay rate			
($1.–$1.99 vs. $2,50)	1. 41	. 47	. 71
($2.–$2.49 vs. $2.50)	3. 57 [4]	. 97	. 39
($2.50+ vs. $1.–$1.99)	—	2. 10 [4]	—
3. Longest job held			
(unskilled vs. highly skilled)	2. 54 [4]	. 80	. 57
(highly skilled vs. skilled)	—	3. 57 [4]	—
4. Local property owner (no vs. yes)	2. 01 [4]	. 78	1. 23
5. Race (nonwhite vs. white)	2. 01 [4]	1. 49	1. 34

[1] Comparison of 59 cases dying within 3 years of retirement with 220 controls.

[2] Comparison of 51 cases dying within 4–5 years of retirement with 220 controls.

[3] Comparison of 52 cases dying within 5 years of retirement with 104 controls.

[4] Relative risk exact 95 percent confidence interval (one-tailed) exceeded 1.00.

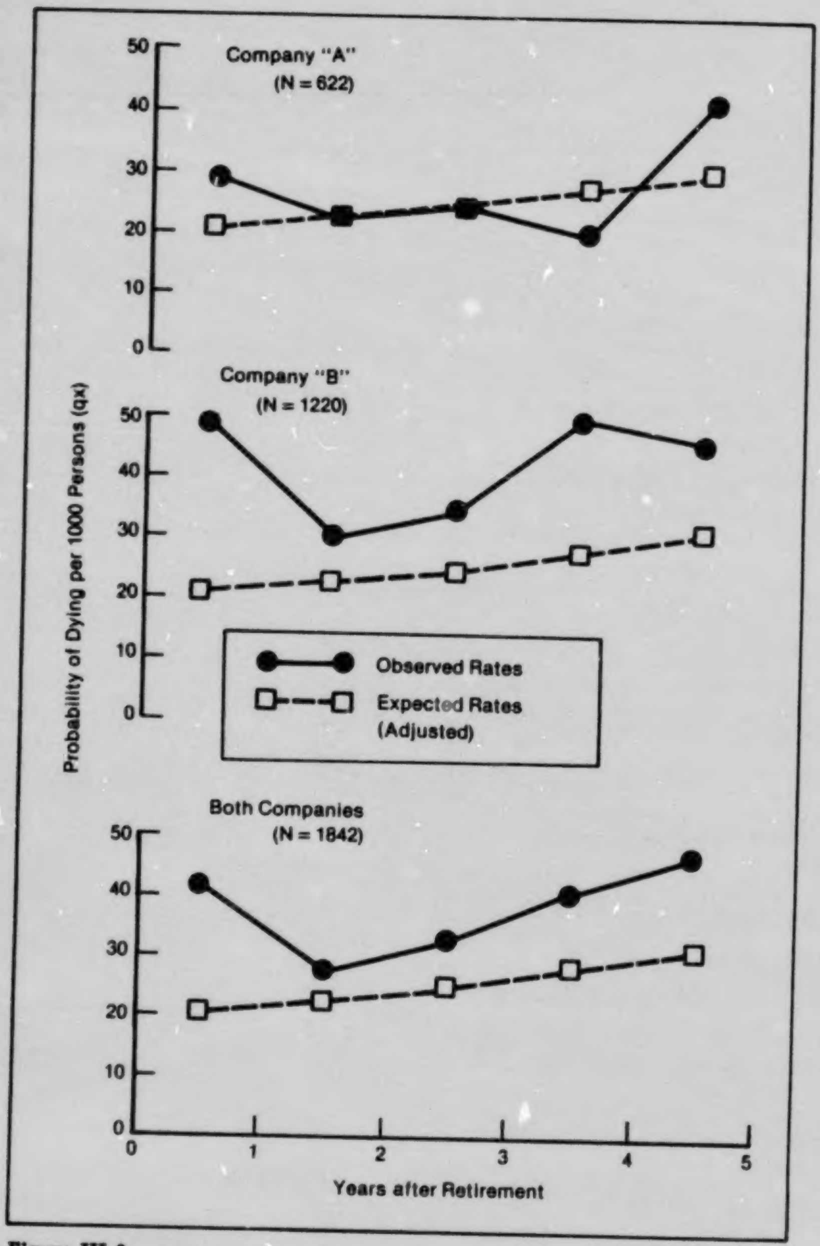

Figure III-9.
Mortality rates after early retirement.

status workers were consistently more likely than higher status workers to die within 3 years of retirement for all six comparisons shown in the normal retirees, group I column. For example, those who had performed predominantly unskilled work were at 2½ times greater risk of early death than were highly skilled workers.

Unexpectedly, lower status normal retirees were not at greater risk of dying in the suspected troublesome period, 4 to 5 years after retirement. In fact, examination of the figures in the group II column shows that the higher paid and higher skilled workers had a more than twofold risk of dying than did the lower status persons.

In contrast with these two columns of figures for normal retirees, social status measures did not predict survival after early retirement. This is understandable from the data in table III–13 which shows that health status was the most significant predictor of survival for early retirees. Notice that for the first three variables there were consistent, and statistically significant, increases in risk of early death after early retirement. For example, there are twofold to threefold increases in risk for persons with a high index of morbidity and frequent insured sickness absences.

Previous health status (i.e., index of morbidity and number of insured sickness absences) was also a strong predictor of mortality for normal retiree deaths in group I. For example, persons with a high

Table III–13.

Risk of early death after normal and early retirement by previous health status

Variables	Normal retirees		Early retirees
	Group I	Group II	
1. Index of morbidity before retirement[1] (high vs. low)	4.57[2]	2.18[2]	2.86[2]
2. Insured sickness absences during career (2+ vs. none)	2.07[2]	1.69	3.50[2]
3. Uninsured absences before retirement			
(1–6 vs. none)	2.91	.82	1.98[2]
(10+ vs. none)	—	—	2.35[2]
4. Index of job satisfaction—uninsured absences among persons who had a low index of morbidity before retirement			
(1–6 vs. none)	1.00	2.64[2]	—
(6+ vs. none)	3.09[2]	3.37[2]	—

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[1] A low index of morbidity refers to persons with no insured sickness absences and no hospitalizations and no work limitations during the 2 years before retirement. A high index of morbidity refers to persons with any or all of the medical events mentioned above. All medical events occurring "before retirement" refer to the 2 years before retirement.

[2] Relative risk exact 95 percent confidence interval (one-tailed) exceeded 1.00.

index of morbidity before retirement were over four times more likely to die within 3 years of retirement than persons with a low index of morbidity. When the calculations in table III–12 were repeated, controlling for level of pre-retirement morbidity, health status did not explain the observed association of occupation and pay rate with death in this group. Previous health status was also predictive of survival in group II comparisons of normal retirees; but again, the associations of pay rate and occupation with survival remained significant when controlled for previous health.

In order to estimate the importance of job satisfaction for subsequent longevity, a surrogate measure of satisfaction was developed based on medically unexplained absences from work. Empirically, uninsured absences of less than a week's duration have been associated with hostile attitudes toward work, both in our own research at another tire plant (*6*), and in the work of other investigators (*29–31*). From table III–13, we see that uninsured absences among those persons who had a low morbidity index (i.e., no objective medical illnesses) before retirement were significantly associated with mortality after normal retirement, especially in group II comparisons. We can only speculate that this group of retirees was experiencing some dissatisfaction with their working situation, since these absences may also reflect medical episodes.

None of the measures of social support, including marital status, number of dependents at time of retirement, or retiree club membership, were predictive of subsequent mortality in either normal or early retirees.

Comparison of Normal and Early Retirees

Thus far the patterns and predictors of post-retirement mortality have been quite different for workers choosing early as opposed to normal retirement. It is possible that persons holding certain jobs or in certain states of health chose an early retirement rather than working until age 65. The sampling design of our case-control studies enabled us to compare the socioeconomic level and several health indices among normal and early pensioners. This was achieved by combining the cases with the controls, weighting the latter group appropriately.

The proportion of early and normal retirees in various socioeconomic groups or health states before retirement are shown in table III–14. A Chi-squared test for the equality of expected proportions in normal and early retirees was obtained using special variance formulas. These formulas took into account the weighting procedures used to obtain the proportions, as well as the rate of dying in each retiree group (*32*).

We see from table III–14 that early retirees held a different mix of jobs, had more education, and were more likely to be white than normal retirees, significantly so at the $p<0.05$ level. Early retirees were found to hold unskilled jobs more often than normal retirees

Table III–14.
Percent distribution of socioeconomic and health status among normal and early retirees

Variables	Early retirees Percent		Normal retirees Percent
Socioeconomic status:			
Longest job held:			
Highly skilled	30.4		27.9
Skilled	18.2		19.3
Semis-killed	17.3		28.0
Unskilled	34.1		24.8
		$X^2=8.20$[1]	
Local property ownership:			
Yes	64.9		70.8
No	35.1		29.2
		$X^2=1.46$	
Last hourly pay rate (dollars) 1964–68 retirees:			
\$0–1.99	39.7		43.1
2.00–2.49	35.0		34.3
2.50+	35.2		22.6
		$X^2=0.54$	
Education (years):			
None	5.3		11.5
1–8	53.9		53.7
9+	40.8		34.8
		$X^2=6.03$[1]	
Race:			
White	94.0		82.9
Nonwhite	6.0		17.1
		$X^2=13.84$[2]	
Health status:			
Index of morbidity 2 years before retirement:			
Low	51.5		66.9
High	48.5		33.1
		$X^2=8.47$[2]	
Hospitalizations 2 years before retirement:			
None	73.9		80.6
≥1	26.1		19.4
		$X^2=2.15$	
Insured sickness 2 years before retirement:			
None	66.3		73.5
1	28.2		23.1
2	5.5		3.4
		$X^2=2.48$	
Work limitations 2 years before retirement:			
Unlimited	69.4		79.8
Limited	30.6		20.2
		$X^2=5.05$[1]	

Variables	Early retirees Percent	Normal retirees Percent
Uninsured absences 2 years before retirement (per month):		
None	25.5	40.9
≥ 1	74.5	59.1
	$X^2 = 10.21$ [2]	
Total insured sickness absences during working career:		
None	35.3	43.2
1	26.8	28.0
2+	38.8	28.8
	$X^2 = 4.85$	

[1] $0.01 < p \leq 0.05$.

[2] $p \geq 0.01$.

The X^2 test for the equality of expected proportions in normal and early retirees was obtained using several variance formulas. These formulas took into account the weighting procedures used to obtain the proportions, as well as the rate of dying in each retiree group.

(34.1 percent vs. 24.8 percent) and the latter were more likely to hold semi-skilled jobs than the former. No significant differences were found among the early and normal retirees in pay rate or property ownership. The early retirees were found to be in poorer health before retirement than were the normals. Significant differences were observed in three of the seven health status measures, including an index of morbidity, work limitations, and uninsured absences during the 2 years before retirement. Other health comparisons using hospitalizations or insured sickness absences, although not statistically significant, were in the expected direction with early retirees having more illness episodes before retirement than normal retirees.

Of further interest is the health status of workers who remained in the company until the mandatory retirement age of 65. Among these normal retirees, 80 percent were under no work limitation at the time of retirement. During the 2 years before retirement, 19 percent had been hospitalized and 26 percent missed work for over 7 days because of illness. Sixty-six percent of workers forced to retire at age 65 had no hospitalization, no sickness absences, and no work limitations in the 2 years before retirement, i.e., had a low index of morbidity.

Discussion

Our studies indicate that mortality after early retirement is higher than would be expected in a working population and that survival after such retirement is related to previous health status. This finding is supported by studies (*33*) among Civil Service employees retiring before the compulsory age of 70. For attained ages 60 to 66, mortality was

9 to 21 percent higher than expected in that population. The Civil Service retirees did not include terminations from work due to disabling illness.

Our findings also indicate that normal retirement, per se, is *not* obviously detrimental to survival. Since 66 percent of men working until age 65 were in relatively good health before retirement, one would not expect to see an immediate rise in mortality after retirement. Likewise, the honeymoon phase theory suggests that the initial period after retirement is not generally stress evoking (*20*).

However, the greater than expected death rates reported in company A during the fourth year, and in company B during the third year are consistent with a disenchantment phase interpretation. The pooled company rates, though not statistically significant, also follow the expected trend. Given the unavailability of an ideal comparison population, the disenchantment phase effect may be greater than is apparent in this study. Since we were not able to interview the normal pensioners during their retirement, we do not know if they were experiencing adjustment problems during these years. We hope other investigators will test the disenchantment phase hypothesis in other prospective studies.

Despite these qualifications, evidence from at least three other studies supports a disenchantment phase interpretation. Martin and Doran (*34*) found elevations in illness rates 4 to 6 years after retirement among blue-collar workers. Solem (*35*) reported elevated mortality rates during the third year after compulsory retirement at age 70 in Norway. Stokes and Maddox (*36*) have also shown that blue-collar workers experience a substantial decline in satisfaction 3 to 5 years after retirement.

The predictors of mortality after normal retirement are consistent with other studies, but only for deaths within close proximity to retirement. Lower socioeconomic status and poor health before normal retirement were significant predictors of death within 3 years of normal retirement. In general, one would expect unskilled workers (or laborers) to exhibit higher rates of dying than other occupations groups. Kitigawa and Hauser (*22*) have shown that mortality ratios for laborers (1.18) and operators (1.08) exceed unity when comparisons were made to U.S. rates. Our findings are also consistent with the Duke longitudinal study (*23*) which found physical functioning and work satisfaction two of the strongest predictors of longevity among older men.

The finding that higher social status was associated with death 4 to 5 years after retirement is not in concordance with the longevity literature, and could be explained if selective survival had taken place and/or if this subgroup of normal retirees had experienced some "disenchantment" with retirement. Selective survival would take place if a preferentially healthy group of unskilled workers survived the first 3 years of retirement, such that their subsequent survival experience was better than that for existing highly skilled workers. This phenomenon would not explain the slight excess in mortality observed in year 4 in company A. Alternatively, the higher skilled workers could have experienced some "disenchantment" 4 years after retirement, resulting in new illness or exacerbation of existing disease. This interpretation would explain the peak in mortality during the fourth year.

Current thinking suggest that social support is an important moderator of life stress (*37*). Elevated mortality ratios have been found among persons without a marital partner (*22*). In our case-control studies, support measures such as marital status at the end of employment, membership in a union retirees club, and number of dependents at retirement were not predictive of subsequent survival. Thus, being married, having dependents, and having some social activity per se were not protective against mortality. Perhaps more qualitative, i.e., actual love and support from spouse, rather than quantitative measures of social support would have been better predictors.

Of final note were the comparisons of normal and early pensioners. Early retirees were in poorer health than normal retirees, especially during the 2 years before retirement. This suggests that some workers who elect an early retirement in the rubber industry may exercise this form of work termination because of declining health, rather than for the opportunity for extra leisure at the end of a working life. Although the early retirees were better educated than the normal retirees, they were more likely to be found in unskilled jobs. This could be explained by their health condition which might have made them ineligible for better skilled jobs.

Summary and Future Research

We have found that the patterns and correlates of survival among early retirees suggest that this form of work termination is chosen by some, but not all, workers because of declining health. The transition into mandatory retirement at age 65 (with the life stress and need for adaptive change that it implies) was not of sufficient moment to cause an immediate increase in mortality. However, within the boundaries of the blue collar subculture were socioeconomic gradients sufficient to markedly affect mortality 3 to 4 years after retirement. When considering the issue of mandatory retirement, it is important to realize that over two-thirds of factory workers who remained in the industry until age 65 appeared to be in good health before retirement, as measured by the absence of hospitalizations, sickness absences, and work limitations. These data point to the need for actual health evaluations of workers in the seventh decade of life.

We are sympthetic with the views of Butler (*38*), who notes that the right to work is basic to the right to survive, and that, ideally, retirement should be an option based upon the individual's needs and wishes and upon an evaluation of his physical and emotional capabilities to function. One might reconsider the current trend toward forced retirements at the fixed age of 65, as well as concentrating efforts toward the improvement of retirement conditions for these mortality susceptible socioeconomic groups. Since an increasingly greater proportion of today's work force is surviving beyond the age of compulsory retirement, we hope other epidemiologic studies will be undertaken in this area.

Future research into the study of retirement, stress, and disease might address the following questions and concerns:

1. What are the conditions under which retirement may be considered stressful, and how might these conditions be identified?
2. What subgroups of the working population are more susceptible to retirement adaptation problems? Does social support affect the adaptation capabilities of these groups?
3. What are the most appropriate health measures, i.e., morbidity, mortality, etc., for detecting a potential stress from retirement?
4. If there are health-relevant phases in retirement, can we distinguish them by intervals of time after retirement?
5. If certain phases of retirement do cause adaptation problems, how might these problems be identified and prevented?

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Discussion

Brehm: The work that Dr. Joseph Quinn did, looking at the longitudinal retirement history survey data from Social Security, indicates a very close interaction between health status and financial status in the decision to retire early. I am wondering to what extent some of that might help to explain some of your data on health conditions after early retirement in terms of the decision to retire being based on health status.

Haynes: We were not able to ask individuals their reasons for retirement since the study population had already retired or deceased. We do not know what their reasons were for taking an early or a normal retirement. Using some statistical weighting procedures, we were able to compare various health and economic variables among the early and the normal retirees. We found that early retirees were less healthy than normal retirees in the population.

Brehm: Before retirement?

Haynes: Yes, in the 2 years before retirement. Among those taking a mandatory retirement at age 65, 80 percent were under no work limitation at the time they retired. Two-thirds of normal retirees had no work limitations, no hosptializations and no insured sickness absences in the 2 years before they retired. So the normal retiree group might be considered in good health, at least in terms of the health measures available for evaluation.

Brehm: In an earlier study of work-related disabilities done at Social Security—the 1966 survey—we found that of the people who opted for early retirement, i.e., actuarially reduced benefits at age 62, something like 30 percent were approved for disability benefits.

Haynes: We found that 30 percent of the early retirees were under work limitations at the time they retired compared with only 20 percent of the normal retirees. We have suggested that the rubber tire industry might consider giving extra benefits to some of the early retirees, at least to those who are taking this form of retirement because of poor health.

However, distinguishing between early retirees who choose retirement because of poor health and those who are taking it for extra leisure time is difficult. I do have the feeling that some of the early retirees were rejects from disability retirement applications.

Nash: Were you able to control or determine whether or not retirement preparation on the part of these people had anything to do with adjustment?

Haynes: There is no retirement preparation program in the rubber tire industry. We did look at membership in a rubber workers union retirees' club which, of course, was available after retirement. We found that membership did not affect subsequent survival. Then again, one could not become a member if he died within a few years of retirement.

Feinleib: Did you have any measures of encouragement to retire early, such as productivity or other evidence that they were not performing as well as the mandatory retirees?

Haynes: We did not have any indication that they were encouraged by the personnel officer to take an early retirement. In terms of productivity, the only measure we have is work limitations which are a good indicator of whether or not the person can function in the job in which he is working. There are limitations for lifting, walking or working in certain areas of the plant.

In terms of their ability to produce or perform in their job, I cannot answer that. It is a good question. I think the answer would be of interest to Congress in their consideration of the abandonment of the mandatory retirement age. How do you go about determining whether or not a person is able to function on the job where he is currently working?

Waldron: Did you try to compare the early retirees to people with similar health status who kept on working? In other words, did retiring improve their health status compared to those people who kept on working?

Haynes: We compared mortality rates among actively employed workers and early retirees. There were only three ages at which you could take an early retirement: 62, 63, and 64. We considered the mortality rates of actively employed workers at these ages. Since the rates in the actively employed group were based on small numerators, I don't think we were able to make stable comparisons.

Atchley: There is a tendency on the part of people to look at mandatory retirement as involuntary, and I just want to remind everybody that a lot of people who wait around to 65 are not necessarily reluctant to go. They are chomping at the bit in many cases. They look upon the mandatory age as social permission to retire. So that if you wanted to look at the effect of mandatory retirement, you really need to separate out those people form the people who Gordon Streib has called "reluctant" mandatorily retired persons.

Haynes: I think that we are only talking about, at the most, 30 percent of workers who retired at age 65 and who wanted to stay on. That would be my best guess from the satisfaction literature. We do not know what the percentages are in other industrial populations.

Cohen: I wonder if this is not supported by the observation that there seems to be a process occurring that is different in time for lower socioeconomic status persons as compared to higher ones; that is, the first 3 years are different for one group than the other and then the 4 to 5 year patterns are different. Do you have any information on what has occurred in that period of time?

Haynes: The only information is from a study by Stokes and Maddox from the Duke longitudinal cohort which examined satisfaction scores among blue-collar workers. They found that immediately after retirement, i.e., in the first 2 years, the satisfaction scores were high. However, they took a sharp decline 3 to 5 years after retirement. White-collar workers showed the opposite trend. They were initially dissatisfied, then after 3 to 5 years they became more satisfied with retirement.

Shanas: Dr. Atchley made the comment which I had previously suppressed. If you analyze older persons retiring from work in this country, particularly blue-collar workers, you find they are looking forward to retirement. In fact, they are holding on until they are 65 so that they can get all the benefits. That is the sort of thing you are getting now, Dr. Atchley, in your research. And this may not have been so in your sample, Dr. Haynes.

Haynes: I think that still goes along with what we found. You can look forward to retirement, get into it and go through a honeymoon phase. Then all of a sudden you become disenchanted with the circumstances that you find yourself in 3 to 5 years later.

It is not retirement per se, but it is circumstances that an individual is put into when he is forced to retire that are probably important, i.e., income, loss of something to do during the day, and loss of friends.

Shanas: I have data which show this period of disenchantment. In addition, scores on the index of functional capacity also rise during that period and then come down. I wonder whether anybody has tried to do any special studies, using physical examinations to find out what is happening to these people during this period. These may explain the increased death rates. I have found, in the past and also now, an increase in limitations, a decrease in mobility, and an increase in use of doctors in that period.

Haynes: Dr. Atchley has an ongoing study in which he is following retirees and is administering a self-report health questionnaire.

Atchley: It is self-reporting on a number of different levels, activities that people can do and general health status. The health trend question seems to be the nearest one from the point of view of separating people out.

Haynes: What do they say?

Atchley: Most of them say "Staying the same." The ones who say, "It is getting worse," have all sorts of problems.

Brehm: These are self-assessment data. There is a followup on the 1966 survey of disability that was done in 1968 or 1969. The assumption was made that people who had reported themselves as work-limited disabled in 1965 would show a decrement in their capacity to function. Some said they were seriously disabled in 1965, but in 1968 they claimed that they had never been disabled in their lives. They rated their disability in terms of whether or not they could get a job.

Shanas: I know that study. In terms of your study, it has always seemed to me that it would have been possible to get a group like that and have them physically examined in what may be considered the target age to see what you find.

Haynes: We tried to follow our workers after retirement in terms of sickness absences and hospitalizations, but there is a ruling that you can destroy records after 5 years, and the necessary records were not available.

Kovar: The Social Security retirement survey and the Parnes longitudinal study both include health measures. They are not very extensive and they are not examinations, but they both have the self-assessment of health.

Haynes: Self-assessment is usually only 70 percent valid when compared to a physician's examination.

Costa: The other longitudinal studies, the Duke study and the Normative Aging Study in Boston, have actual measures for retirees. Colleagues of mine formed a cohort of 300 people who retired with self-reported health and measures obtained from extensive physical examinations.

Palmore: In the Duke study, we do have physical examinations. The trouble is, we only had 75 people who retired during this study. So far, we have not found any noticeable effects of retirement on before and after measures except that income goes down, there are more hours of leisure, and the retirees interact with people a little less.

Haynes: I think the Duke sample is a somewhat higher status group than the workers we have been looking at. Unless you can separate blue-collar from white-collar workers, you probably won't find an effect.

Brehm: I would like to refer to a report made yesterday involving the distinction between an impairment and a disability. In a study that was done using the 1970 Census data, which for the first time asked specific questions about the extent of work-related disability, we did an aggregate analysis, looking at the percentage of people within a state who define themselves as work-related disabled. You can account for something like 80 percent of the variation among states by data on the level of poverty, and conditions of the labor force. The fact is that these measures did not produce impairment to health status in the population in general.

Now, how you explain this is difficult. At one point, you are asking someone how well he performs, rather than dealing with the issue of what is wrong with him. We know there are people in society who have all kinds of health conditions or impairments who do not define themselves as disabled. Early retirees, we presume, are opting out of the labor force, so they just become or accept being disabled, at least psychologically, if not in the medical sense. It is very complex at this point.

Kannel: In the Framingham study we have a cohort which now has an average age of 65 years and could be studied. Many of these people are going through retirement; a good number have already done so. We have records of systematic routine measurements of health. We know about hospitalizations. We know about interim illnesses. We monitor admissions to the only hospital in town daily. The information is available for some retirement studies.

Cohen: Do you also ask about appraisal of health status?

Kannel: That is being done. One could, at the same time, determine how people's perception of disability relates to what is actually found on the measurement.

Haynes: One item that should be collected in any study is the exact date of retirement in order to replicate the analysis that we have done. This is important if you are looking for a subtle effect, say 3 or 4 or 5 years after retirement. A lot of studies do not record this information. It is probably a simple thing to do.

Atchley: It depends whether you are studying an employer centered operation or a general population. There are a lot of people who cannot tell you the exact date that they retired.

Shanas: You can try to get it two ways. How old were you when you retired and how long ago? What is the year that you retired? Sometimes the two answers will balance out.

Kasl: A few comments—one on the issue of self-reported health. I think there are some data which show by comparison of self-reported with physical examinations that those with a strong commitment to the work role are the health optimists. One might assume that reluctant retirees on self-report are health optimists.

From the literature on the blue-collar workers, I find that they seem to have such a low commitment to the work ethic. The reluctant retiree, aside from financial considerations, is an unusual kind of a person and the interpretation has been made strictly in terms of a commitment to a work role. I think in the future it might be interesting also to look at his social environment. It may not be that the work role is so attractive, that he may be more of a social isolate in his social life that makes him a reluctant retiree. It is the difference between the approach motivation and motive motivation. The work is not attractive, and he is trying to work something else. I think that might be a real requirement.

Siegel: I would imagine that studies involving retirement, particularly of white-collar people, will become increasingly difficult since the concept of retirement is becoming shady. We have not only second careers but also part-time, trial, and gradual retirement. For example, there is the Federal Government retiree or the military retiree who goes back to work either part-time or full-time. There is emerging a different concept of retirement among older people.

Atchley: There is a measurement problem just in terms of relative work loads. For example, if you take professors and you ask them how many weeks they work on the average and you plot that by age, you see that on the average professors start to retire when they are around 40. Their average work week starts going down in terms of their estimates of the amount of time they spend related to the job.

There is an interesting thing here, though. If you want to learn the extent to which an individual is retired, you should ask the retiree what the normal work load is in his occupation and what the normal number of weeks of work is in that occupation. Then find out how many weeks he works in relation to the normal work week and what the normal work load is in the normal week's work in his occupation. It is not a simple matter to decide how much of a work load in his occupation an individual is carrying if he is partially retired. Fortunately most people retire completely, so practically it is not that much of a problem.

Effects of "Involuntary" Relocation on the Health and Behavior of the Elderly*

Stanislav V. Kasl, Ph. D., Adrian M. Ostfeld, M.D., Gerald M. Brody, M.D., Laura Snell, M.P.H., and Christine A. Price, M.P.H.

Department of Epidemiology and Public Health
Yale University School of Medicine
60 College Street
New Haven, CT 06510

Recent studies in psychosomatic medicine and social epidemiology, which also fall into the broad category of "social stress and disease," have been heavily concerned with *social change* and *life events* (*1–3*). This is probably because events of social change are most likely to encompass one or more of the major meanings of "stress": threat, uncertainty and unpredictability, conflict, excessive demands, failure of aspirations, frustrations of needs, and/or loss. Investigators such as Cassel (*4*), Groen (*5*), Hinkle (*6*), Holmes (*7*), Kagan (*8*), and Rahe (*9*) have all assigned great significance to social change in the etiology of illness.

Much of this work is being subjected to careful critical scrutiny (*10–14*) and certain conclusions are becoming, in our opinion, increasingly more compelling: (1) Prospective study designs are particularly valuable and desirable; (2) The intensive study of one social event at a time (bereavement, retirement, job loss, and so on) is much more likely to advance our knowledge than continued reliance on the schedule of recent experience type of approach (*7*, *9*), in which an inadequate self-report instrument lumps together a variety of past experiences in one total score; (3) A richer theoretical framework is needed for the study of health effects of social change: this framework must focus not only the experiential aspects of the change, e.g., magnitude, duration, unpredictability, novelty, extent of control, and the like, but also the social-environmental or ecological aspects, e.g., social networks and social supports, occupational environment and job demands, residential settings, and so on.

One of the life events which has received a good deal of attention is *residential change* (*15–17*). The kinds of studies which fall in here have dealt with: moves to the suburbs, moves from "slums" to public housing, change from community living to institutional living, institutional transfers, relocation because of urban renewal, and moves to specialized settings such as elderly housing and retirement communities. In general, such studies of residential change have had one of two broad objectives: (1) To study the impact of a certain residential environment or some dimension of it; for example, to assess the differ-

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ential effects of "good" vs. "bad" housing, one may study, in classical experimental tradition, people who move to better housing. The emphasis is on determining the environmental impact, not on studying the change; (2) To study the effects of the experience of changing residence; the emphasis is on the change itself and any interest in how the residential environment was before and after the move is secondary or nonexistent.

Studies of purely voluntary individual residential moves, such as to the suburbs, have generally failed to demonstrate convincingly any consequences of the moves which could not be interpreted as "intended" changes, i.e., the reasons for which the move was made in the first place—increased satisfaction with housing, increased social life, etc. (*16–19*) In other words, such residential moves are so firmly embedded in life cycle variables, in goals, aspirations, plans, intentions, and perceived opportunities, that any causal inferences about effects are exceedingly difficult.

Studies of the effects of improvement in housing conditions on health and behavior, such as the well-known Baltimore study (*20*), have generally not been able to show any significant benefits, though they do reveal some changes in residential satisfaction (*16*, *17*). It is quite likely that the possible benefits of improved housing are being masked by possible adverse effects of the residential move itself, particularly if it represents a profound social uprooting experience. That is, the residential change, which has improved the housing, may also involve: (1) segregation from friends and relatives, leading to reduced social contacts and mutual help and support; (2) unfamiliar surroundings and unknown requirements; (3) new schools, changes in police protection; (4) greater unavailability of certain facilities. The recent experience with poorly planned high rise public housing (*21–23*) may be a particularly striking illustration of how the possible benefits of improvements in certain housing parameters may be overwhelmed by the negative effects due to the social context factors.

The study of involuntary residential moves offers the greatest promise of being able to detect and assess adverse effects on health and behavior. The involuntary aspects of the move, such as in urban renewal or institutional transfer, tend to minimize the potential confounding and bias which come from various self-selection factors. Moreover, many such moves do not involve major changes in the quality of housing, thus reducing the possibly complicating effects of the environmental factors. Finally, the imposition of a residential move on persons who do not seek it (and probably consider it unwanted and undesirable) suggests that one is selecting for study the more severe and traumatic end of the spectrum of the residential move phenomena.

Many of the studies of involuntary residential change deal specifically with the elderly. This may be both because this demographic subgroup is more frequently exposed to such experience, and because they are more likely to be more vulnerable to the adverse effects of it

(*24, 25*), thus allowing for a more powerful test of the hypothesis. The research literature on involuntary relocation of the elderly was reviewed in 1972 (*15*) covering studies through 1970. Many additional studies have appeared since then (*26–32*). The results of these studies are difficult to summarize inasmuch as a consistent and coherent picture does not emerge. However, one conclusion is inescapable: the simplest form of the hypothesis, namely that there will be increased mortality and morbidity among elderly experiencing involuntary residential relocation—irrespective of the social context of the change and the way it is handled, and/or irrespective of the nature of the before-after difference in the residential environment—is unlikely to be supported and is not worth testing. It is too simplistic given the complexity of the phenomenon and by itself will not increase our understanding.

On the other hand, there is a good deal of evidence which points to specificity of circumstances under which adverse health effects may be found. For example, relocation of elderly from one institution to another may lead to increased mortality, but this is totally preventable with careful casework service and psychological support. Similarly, healthy elderly may not show any effects of the relocation, while those who are physically or mentally impaired may be the ones who will show the adverse health effects. (But since such impaired individuals are at greater risk of poor future outcomes under any circumstances, proper adjustments must be made or suitable comparison groups found.) Other evidence, most of it indirect, would also suggest the following characteristics of elderly who may be at increased risk for adverse effects: being male and older; living alone and having few contacts with friends and kin; being in poor financial circumstances, of lower social class, and with lesser access to various social services; having lived in the old neighborhood a long time; low in morale and life satisfaction; reacting to the move with depression, with a feeling of giving up, and hopelessness-helplessness.

It is also necessary to pay closer attention to the phenomenon we wish to study. The implicit paradigm for the studies of involuntary relocation is Fried's work "Grieving for a Lost Home" on urban renewal (*33*): elderly residents in an old, intact neighborhood, where they have lived for many years and where rich and strong social ties exist; then the elderly residents are forced out. They receive no help with relocation; they scatter throughout the city, finding available housing which is seldom as good as where they had lived previously, and generally costs more. But can this phenomenon still be studied? It is likely that the following picture is more current: the elderly residents are already somewhat social isolates, living in neighborhoods which are undergoing various piecemeal changes, usually for the worse. The residential move, though forced on them by various circumstances, may be welcomed by many of them. They may receive assistance with the move and they generally go into federally subsidized elderly housing, which in fact represents a residential improvement without the usual financial penalty. Certain services may be more readily available, e.g., a medical clinic on the premises, and the potential for new friendships with other elderly may be great.

In short, the challenge in future studies of health effects of relocation on the elderly is to collect enough information on (a) the relocation experience itself, both objective data and subjective perceptions; (b) on the individual characteristics of the relocatees; and (c) on the social concomitants of the change, so that we may begin to understand why some elderly improve in their health, others deteriorate, and still others show no change. Lawton and Yaffe (*34*) have shown that elderly relocatees, compared to controls, had more changes in health status both for the better and for the worse. Now we need to study the determinants of such differential outcomes.

The general literature on housing and health (*15–17*) would seem to suggest that the elderly as a group are more susceptible to both the benefits of improved housing and the adverse effects of the social uprooting experience accompanying residential change.

Methods

A prospective inquiry was conducted into the behavioral, psychological, and health consequences of involuntary relocation in the elderly. Four sets of variables were assessed: health, psychological state, physiological data, and social behavior. Data were collected on four occasions over 2 years on 225 elderly people who were forced to move and 173 from the same neighborhoods as the cases who had not moved. Interviews on cases (relocatees) were scheduled as close to the date of the move as possible, and at 3 months, 12 months, and 24 months later. Controls were interviewed at comparable intervals.

The cases were all persons in New Haven, Hartford, and West Haven, Connecticut, age 62 and over, who were forced to move within a specified time period because of urban renewal, eviction, condemned or substandard housing, fire, and extreme financial hardship. Their names were obtained by systematically searching the records of the various relocation agencies in the three cities. Excluded from the study were those elderly who: (a) moved for reasons of poor health; or (b) moved outside of administrative channels and did not come to the attention of the relocation agencies. All cases moved to federally subsidized housing for the elderly. Among the cases eligible for study, 79 percent agreed to participate.

As stated above, the controls were persons who came from the same neighborhoods as the cases. They were identified through a systematic process which called for enumerators to go to specific block groups and, using predetermined procedures, to enter selected houses and enumerate elderly residents eligible for study. The controls were group matched with cases on: age, sex, race, and marital status. This method of selection (neighborhood control) also yielded controls comparable on social class. Of the controls approached for study, 64 percent agreed to participate.

A random sample of control refusals was contacted somewhat later with a request to provide limited information and 90 percent complied. Analysis of this information revealed no significant differences from participants on the usual sociodemographic characteristics or on prevalence of grave illness.

All interviews were conducted in the residences of the subjects by trained interviewers. The interview schedule was totally structured and of considerable length. On the average, it took about 90 minutes to complete. Formal reliability studies on the medical portion of the interview and medical diagnoses were carried out. The interviewers (after their training) and a physician collected the relevant data double blind and in a randomized order on 26 elderly persons who were not part of the study. The agreement on presence or absence of the 16 diagnostic categories used in the study was 94 percent.

It is important to recognize the limitations of the study. To begin with there is a certain vagueness regarding the boundaries of the phenomenon being studied. As noted above, one can rarely study involuntary relocation in its purest form these days with urban renewal or highway construction destroying an old, but intact and viable neighborhood from which everybody has to move. What we did study was relocation among poor elderly who were moving under a greater variety of labels such as redevelopment (26.2 percent), eviction (9.3 percent), financial hardship (32.9 percent), Old Age Assistance (24.4 percent) and miscellaneous others or combinations of above (7.2 percent). It may be suspected that these labels hid a good deal of variation in the extent to which the move—and its timing—was forced upon the individual. For example, a landlord who raises the rent "forces" the elderly person on fixed income to move out; but it may be the second raise which did it and the decision might have been facilitated by the elderly person's knowledge that subsidized elderly housing recently became available. There is no way to overcome these problems, other than by collecting additional data from the respondent regarding the circumstances of the move (how voluntary, planned, desired, etc.) and analyzing outcome data in relation to this information. Such data were collected in our study.

A problem related to the vagueness of the boundaries of the phenomenon is our inability to determine precisely how completely we identified all individuals eligible for study; that is, how many elderly, experiencing a relocation under comparable circumstances, escaped the notice of a relocation agency. Discussions with the housing authorities and a search of demolition records suggested that this group could have been as large as 30 percent of all elderly involuntarily relocated in that time period.

The definition and selection of controls also represented somewhat of a problem. Essentially, we wished to find elderly persons who: (a) were living in the same neighborhood and in comparable housing, and (b) who were similar on the major sociodemographic characteristics, but were not going to be relocated. Table III–15 indicates the extent to which we were able to succeed. As can be seen, the controls are different on a number of interrelated variables: More are still married (hence more males); and more own their homes, and their income is higher. In retrospect, these differences were inevitable, i.e., if one goes to a particular block group in a deteriorating area of a city, those elderly who are not moving out are more likely to live in privately owned homes, are more likely to be still with spouse, and are likely to be somewhat better off financially. Table III–15 reveals that cases and controls are quite comparable on a number of other demographic variables, such as race, place of birth, age, education, and religion. It must be noted that the income differences between cases and controls are not as large as they appear to be. This is because for 28.9 percent of the controls vs. only 10.7 percent of the cases, the reported income is for two people. Table III–15 also shows the results with two subjective indicators of financial status; both measures show much smaller differences between cases and controls than does the objective monthly income information.

Cases and controls were compared on various indicators of health status and medical care. These data are presented in table III–16 which were collected at the initial interview. An indication of the assessment of those variables needing further detail is as follows:

Table III–15.

Social and demographic characteristics of relocated elderly cases and controls

	Cases (N=225)	Controls (N=173)
Sex—percent males	28.4	38.1
Race—percent non-white	24.4	26.6
Place of birth—		
Percent in Conn	32.0	32.1
Percent non-U.S.	29.5	21.8
Age—mean and S.D.	71.9 (±6.4)	72.2 (±6.9)
Education—percent with some h.s. educ	34.9	41.9
Religion—percent Catholic	51.7	52.6
Current marital status—		
Percent married	19.6	35.3
Percent widowed	54.7	41.0
Home ownership—percent own home	2.7	27.2
Monthly income [1]—		
Median	$190.0	$250.0
Percent > $200	35.3	62.2
Financial status at end-of-month—percent "poor" [2]	23.7	15.0
Difficulty living on income—percent "Difficult" [3]	31.2	23.5

[1] For 10.7 percent of cases and 28.9 percent of controls, this is income reported for two people.

[2] Those choosing alternatives "using up savings" or "going into debt."

[3] Those indicating "it is very difficult" or "it is just about impossible."

Angina pectoris: Based on questions from the Framingham study; the inquiry deals with chest pain, its location, duration, antecedent circumstances, and its disappearance upon resting.

Myocardial infarction: Based on answer to "Has a doctor or nurse ever said that you had a heart attack?"

Intermittent claudication: Based on questions concerning cramps or pain in legs, related to walking, and disappearing within a couple of minutes after respondent begins to rest.

Stroke: Based on several questions which reveal acute onset of a neurological deficit, compatible with a vascular lesion; doctor indicated the trouble was "stroke."

History of diabetes: Based on answer to "Has a doctor or nurse ever said that you had—sugar in your water?—sugar in your blood?—diabetes?"

Table III-16 shows that a remarkable similarity exists between cases and controls on the various indicators of health status. The two groups are comparable on the several items of health history, on several aspects of medical care, and on diverse indicators of global self-assessed health status. The last item, "subjective age" has been used in many studies of elderly as the most general single item indicator of perceived health and well-being.

Table III-16.

Comparison of cases and controls on diverse indicators of health status and medical care, based on data at initial interview

	Cases	Controls
Angina pectoris—percent possible or probable	7.9	7.0
Myocardial infarction—percent possible	13.5	13.0
Intermittent claudication—percent present	4.4	6.8
Stroke—percent possible or probable	11.9	7.0
Prior C.V. disease—percent scored positive on one or more of the above four	27.1	26.7
History of diabetes—percent positive	13.0	15.7
History of high blood pressure—percent positive	41.5	39.4
Current medication for high blood pressure—percent positive	26.3	30.0
Hospitalization previous year—percent "yes"	19.5	17.4
Saw doctor previous year—percent "yes"	79.8	77.6
Activities given up because of health—percent "yes"	35.3	32.9
Rating of "health today"—percent "fair" or "poor"	32.3	32.5
Rating of "health compared to people of same age"—percent "same" or "somewhat worse", or "much worse"	30.4	31.7
Subjective age—percent viewing self as "young" or "middle-aged"	56.8	56.1

Results and Discussion

These findings must be viewed as preliminary since they have not yet been subjected to more probing analysis (such as controlling for many relevant variables) and to various systematic checks on the relationships. Second, because of severe limitations of space, the choice of what to present from a large pool of variables collected longitudinally is particularly difficult. Since we have neither carried out in-depth systematic analyses of a few outcomes, nor have we skimmed the data for the "best" or "most interesting" results, we can only present a sampling of findings which may be somewhat haphazard. The intent is to present a variety of measures and results in order to convey the theoretical orientation behind the study design and the direction we shall be taking in analyzing the data.

Table III–17 represents some perceptions and evaluations of the move among the relocatees. These data strikingly illustrate that the majority of the relocatees considered the move voluntary, desirable, and expected improvement in their lives after the move; they did not anticipate missing the old neighborhood and the people in it, and expected a quick resettlement. However, the move was seen as a big change and there was some worry and uncertainty about it. Overall, it is clear that the process for identifying cases did not yield a great proportion of elderly who saw the move as forced upon them and as undesirable, and who would be leaving the old neighborhood with great regret. The process identified cases where external circumstances eventually precipitated the move, or affected its timing, but this is far from saying that the move was seen as involuntary. We are still confident that we are examining a significant social change in a vulnerable subgroup (poor elderly), but it would be foolish to believe that the change was uniformly stressful for all relocatees and that adverse effects on health and well-being should be predicted across the board. The data in table III–17 will be combined to create a conditioning variable which (in later analyses) may be expected to modify strongly the average group effects.

A number of the questions listed in table III–17 were asked again at followup interviews. It is interesting to note that 3 months after the relocation, the distribution of answers was substantially the same as shown in table III–17. The biggest difference—and not a large one at that—was on "miss the people in the neighborhood": 44.1 percent indicated "very much" or "somewhat," compared to the 33.6 percent at initial interview.

Table III–18 offers some information about the cases' and the controls' perceptions of the neighborhood and of the apartment, collected at initial interview and 1 year later. The data on perceptions of the neighborhood reveal the following: (1) Controls do not show any significant changes over time (examined only on those for whom data at both interviews were available); (2) At initial interview, there are two significant differences between cases and controls: Cases describe the neighborhood as somewhat less friendly and they are clearly revealing

Table III–17.
Some perceptions about the move among cases—initial interview

	Distribution of responses among cases (in percent)
Voluntary vs. forced move:	
Completely forced, no choice	14.0
Largely forced, some choice	10.2
Somewhat forced, good deal of choice	12.4
Wanted to move, choice respondent's	63.4
How much looked forward to new move:	
Very much	67.0
Somewhat	14.8
Very little	5.5
Not at all	12.6
How worried about moving:	
Very much	24.0
Somewhat	22.9
Very little	13.1
Not at all	40.0
How much know what to expect, what it will involve:	
Know very well	36.1
Have some idea	32.0
Know very little	14.2
Don't know at all	17.8
How much will miss the neighborhood, that part of the city:	
Very much	10.6
Somewhat	12.8
Very little	9.0
Not at all	67.6
How much will miss the people in the neighborhood:	
Very much	14.1
Somewhat	19.5
Very little	13.0
Not at all	53.5
How long before feel at home and settled in new place:	
A week or so	68.6
About a month	20.1
A few months	7.1
Around half a year, probably never	4.1
How much of a change in life is this move:	
Hardly any change	19.1
A small change	11.0
A fairly important change	19.7
A very big change	34.7
It will change my whole life	15.6

Continued

	Distribution of responses among cases (in percent)
Will change life for better or worse:	
Very much for the better	53.3
Somewhat for the better	32.3
Things will stay about the same	10.8
Somewhat for the worse	1.8
Very much for the worse	1.8

their desire to leave; (3) Cases show a number of significant changes over time: The new neighborhood is seen as more friendly, cleaner, more pleasant, with more friends, changing less, and more of place where they want to stay; (4) Inspection of data on cases collected at 3 months after relocation reveal that all but one of the changes seen at 1 year were evident at 3 months: The one difference is on "full of friends," where at that time 37.6 percent of cases choose this response, a value intermediate between initial interview and 1 year later.

The data on the perceptions of the apartment reveal the following: (1) Controls show no significant changes over time. (2) At initial interview, cases describe their apartment significantly more negatively on all dimensions except "big enough." (3) Cases show significant changes over time on all dimensions but "big enough." (4) Inspection of data on cases collected at 3 months after relocation reveals almost identical percentages as those seen for 1 year later.

Overall, the data in table III–18 suggest that cases and controls had initially fairly comparable evaluations of their neighborhood, but cases had more negative evaluations of their apartment. Over time, cases revealed much more positive evaluation of their new neighborhood and their new apartment; these changes took place rapidly and were evident by 3 months after relocation. There is no doubt, then, that the move was seen as a substantial improvement in the residential environment.

Individuals who have a more negative evaluation of their neighborhood and apartment are, understandably enough, more ready to indicate that they want to leave. However, the differences between cases and controls on this variable at initial interview are so large that the cases' somewhat more negative evaluations of the neighborhood and the apartment alone cannot account for them. The best guess is that: (a) The term "want to" is somewhat ambuguous (e.g. "I want to leave because I can't afford the apartment") (b) The cases knew they were about to move and this could have led to some implicit reevaluation (reduction of cognitive dissonance) about how much they wanted to leave. The notion of cognitive dissonance influencing perceptions would also apply

Table III–18.
Some perceptions of the neighborhood and the dwelling unit, at initial interview and 1 year later

	Initial interview		1 year later	
	Cases (N=183)	Controls (N=164)	Cases (N=146)	Controls (N=104)
Percent describing the *neighborhood* as:				
Friendly (vs. unfriendly)	59. 9	73. 7	78. 9	73. 2
Safe (vs. dangerous)	43. 6	39. 3	53. 2	53. 4
Clean (vs. dirty)	54. 9	56. 1	81. 4	62. 7
Pleasant (vs. unpleasant)	63. 4	66. 5	83. 5	74. 3
Full of friends (vs. full of strangers)	29. 1	29. 3	48. 9	33. 0
Staying the same (vs. changing)	46. 2	38. 1	63. 9	49. 5
A place where you want to stay (vs. leave)	28. 7	63. 5	84. 0	68. 7
Percent describing the *apartment/house* as:				
Comfortable (vs. uncomfortable)	77. 5	92. 0	93. 2	93. 3
Pleasant (vs. unpleasant)	78. 7	88. 8	93. 2	91. 3
Big enough (vs. too small)	82. 0	89. 6	84. 2	88. 5
Safe (vs. dangerous)	67. 2	79. 2	93. 0	81. 4
Where you feel at home (vs. don't)	69. 8	84. 1	87. 7	84. 6
A place where you want to stay (vs. leave)	29. 3	69. 1	85. 6	74. 0

to table III–17, where the results may overestimate the extent to which the move was voluntary, desired, and looked forward to.

Table III–19 presents data relevant to the general question: How were social networks and social activities changed by the relocation? Comparison of cases and controls at initial interview reveals the two groups to be fairly similar. The significant differences are: (a) Cases know fewer friends to call on, and (b) Cases are less likely to have a "confidant" who lives in the same building or the same neighborhood. Adjusting for marital status differences between cases and controls reduces the first but not the second difference to insignificance (about one-third of married respondents list their spouse as the "confidant"). Changes over time reveal: (a) Cases feel they know more neighbors well enough to call on, but actual visiting with neighbors shows little change; (b) The percent of cases who report no close friends goes up somewhat, and actual visiting with friends goes down even more noticeably (however, since controls are also showing some changes, the differential changes between cases and controls are not significant); (c) A direct inquiry regarding wanting more social interaction reveals no differential case-control changes; and (d) The "confidant" data show one striking change—about 20 percent more cases after relocation report that their "confidant" lives in the same building. However, frequency of contact with "confidant" doesn't go up much.

On balance, these results do not provide support for the notion that the relocation represents a social uprooting experience with a consequent diminution in social contacts and interaction. Inspection of results for the 3 months followup interview reveals findings which are generally intermediate between initial interview and 1 year followup (e.g., 20.8 percent of cases with "confidant" report he/she lives in the same building) and thus do not alter our general conclusion.

The bottom of table III–19 presents data for three common activities which among the elderly are considered to be indicative of morale and well-being. The cases reveal a mild consistent trend toward a somewhat lower activity level, but it is not large enough to be reliably different from controls.

We have also examined other measures that reflect more directly the mental health and well-being of the elderly. One is an index of life satisfaction, based on the Neugarten scale (*35*). This measure proved totally insensitive to the relocation experience: Cases and controls were practically identical at initial interview, and thereafter the two groups showed minimal fluctuations over time.

We have also used an abbreviated version of the Multiple Affect Adjective Checklist (*36*) and derived indices for anxiety, hostility, and depression. Again, we are not impressed that there were any notable effects of the relocation experience on affective states. The results so far suggest the following reliable but small effects: (1) Cases show some drop in anxiety (initial to 3-month interview), hostility (initial to 1-year interview), and depression (initial to 2-year followup). (2) Controls show nonsignificant fluctuations and very small trends upward over time.

Table III–20 presents the data on selected outcomes. Mortality data do not reveal a significant difference between cases and controls; in fact, controls have a slightly higher rate, against expectation. Deceased subjects were slightly older at initial interview than survivors (74.3 vs. 71.8, $p<0.02$, respectively). Moreover, those with prior history of cardiovascular disease, had a somewhat higher mortality than those without such history (15.5 percent vs. 10.8 percent, n.s.); however, neither age nor history of C.V. disease are related to case vs. control classification (tables III–15 and III–16). Marital status and monthly income, two variables on which cases and controls are different, were not related to mortality.

Nursing home admissions are significantly greater among the cases during the 2 years of followup. To be sure, this is a complex "health status" outcome variable and proper statistical controls (e.g., adjustments for marital status and income) have not yet been run.

Table III–19.
Social networks and social activities, at initial interview and 1 year later

	Initial interview		1 year later	
	Cases (N = 183)	Controls (N = 164)	Cases (N = 146)	Controls (N = 104)
Number of neighbors knows well enough to call on:				
Percent with none	35.5	29.2	20.0	22.9
Percent with 4 or more	18.8	31.5	37.4	36.2
How often do your neighbors visit: Percent "never"	43.5	38.8	41.1	38.8
How often do you visit your neighbors: Percent "never"	44.3	50.6	47.9	52.1
How many people do you consider close friends:				
Percent with no close friends	16.1	13.2	23.5	12.6
Percent with close friends but not in neighborhood	33.5	31.0	29.6	34.0
How often do the friends come to visit:				
Percent "never"	25.0	28.2	41.9	32.4
Percent with once a week or more	43.7	37.6	31.0	35.2
How often do you visit the friends:				
Percent "never"	32.0	32.4	49.0	42.9
Percent with once a week or more	33.5	30.0	24.2	28.6
Frequency with which R would like to get together with people R enjoys—percent wanting more	35.3	28.8	29.4	22.5
Percent with "confidant" [1]	90.4	89.9	86.7	38.7
How often see "confidant"—percent with at least once a week (for those with "confidant")	69.1	70.4	72.0	68.4
Residence of "confidant" (for those with "confidant")				
Percent, same building	12.4	22.8	32.8	20.5
Percent, same neighborhood	15.7	28.3	11.5	33.3
Percent, same city	36.6	26.0	23.0	24.4
Percent, elsewhere	35.3	22.8	32.8	21.8
Daily activities:				
Taking a nap—percent "never"	47.4	43.8	39.7	46.2
Sit around and do nothing—percent "never"	63.4	67.7	59.5	58.1
Take a walk—percent "everyday"	46.8	43.5	37.9	47.9

[1] Based on the question: "Is there one person that you feel particularly close to—that is, somebody that you can be completely yourself with and in whom you have complete trust and confidence?"

The hospitalization data reveal a significantly greater rate for cases than controls during the first year; the second year difference is in the same direction but somewhat smaller. Supporting these results are the findings on rates of "severe" hospitalization (i.e., involving an operation) and mean number of hospitalizations during 2 followup years. Additional analyses were also run in which cases and controls were further stratified on marital status, income, and previous history of C.V. disease. Combining the comparisons according to Cochran's method (*37*) revealed significance levels virtually identical to those in table III–20. Moreover, there was no evidence of any interactive effects between case vs. control and any of these three stratification variables in relation to the outcome, hospitalization.

Incidence of new disease was examined only among respondents who at initial interview were free of any evidence of the disease. Table III–20 reveals a higher incidence of stroke, angina pectoris, and intermittent claudication among the cases; two of these differences are of borderline significance.

The data on physician visits reveal a somewhat higher rate of visits among the cases, which is significant for the first followup year. However, since the rate of visits among the controls is unusually low, compared to initial interview (table III–16) or the second followup year, we do not have much faith in this significant difference. Incidentally, the vast majority of cases maintained the same source of medical care after relocation, so this is probably not an additional source of confounding effects.

The last variable in table III–20 reveals a modest change in self-assessed health status: Cases show a slight change toward evaluating their health as poorer at 1 year followup compared to initial interview, while controls show a tendency to rate their health somewhat better over the same time period. Stated another way, 20 percent of cases and 38 percent of controls improved in their perceived health status, while 35 percent of cases and 26 percent of controls went down in self-assessed health.

Overall, the results in table III–20 provide reasonably good support, on a broad set of indicators, for the notion that cases going through relocation experienced somewhat more adverse health outcomes than controls who remained residentially stable. In the near future we shall be examining various descriptors of the relocation experience (e.g., table III–17) in relation to the severity of the adverse health outcomes.

We have also begun analyses of various biological variables, especially—blood pressure, serum cholestrol, serum glucose, and serum uric acid. A first, crude examination of means and changes over time leaves us quite unimpressed: The values remained pretty stable, and cases and controls remained comparable throughout. The only exception was a lower mean serum glucose (casual blood sugar) among the

Table III–20.
Selected health outcomes among cases and controls

	Cases	Controls	Significance
Mortality for 2 years of followup—percent deceased	10.2	13.9	n.s.
Nursing home admissions for 2 years of followup—percent admitted	6.2	2.3	0.04[1]
Hospitalization experience:			
Percent hospitalized during 1st year of followup	27.8	12.7	<.01
Percent hospitalized during 2nd year of followup	27.1	18.2	n.s.
Percent hospitalized during 2 years of followup	30.7	19.1	<.01
Percent hospitalized who also had an operation, during 2 years of followup	16.5	8.6	<.05
Mean number of hospitalizations, during 2 years of followup	.64 (±.96)	.38 (±.83)	<.005
Incidence of new disease:			
Stroke—percent possible or probable	16.9	9.4	.06[1]
Angina pectoris—percent possible or probable	11.6	4.9	.08[1]
Intermittent claudication—percent present	11.7	9.3	n.s.
Visits to a clinic or doctor's office:			
Percent who made visit(s) during 1st year of followup	83.8	72.2	<.05
Percent who made visit(s) during 2nd year of followup	89.4	83.3	n.s.
Rating of "health compared to people of same age": Mean change in rating between initial interview and 1 year followup (in standard scores; negative change means health at followup rated worse)	−.18	.22	<.005

[1] Based on Fisher's exact test.

cases at initial interview. This difference is compatible with the interpretation that usual food habits of cases at the time of relocation were temporarily disrupted or altered.

We have also begun analyses of health outcomes in relation to various psychosocial variables. From table III–21 it is evident that interviewer's rating of respondent's affective status is predictive of subsequent mortality. These ratings are not correlated with age, monthly income, or prior history of cardiovascular disease; married respondents are somewhat more likely to be rated as "happy," but martial status was not associated with subsequent mortality. The association between interviewer rating and subsequent mortality was virtually identical among cases and controls, providing no support whatever for the hypothesis that the stressful experience of relocation would enhance the predictive effect of the ratings of depression.

Table III–21 indicates some support for the notion that elderly respondents who are more likely to be social isolates (as measured by the question about friendships with neighbors) have a higher mortality rate. Again, this social predictor was not correlated with age, monthly income, marital status, and history of prior cardiovascular disease. Furthermore, no interaction between case vs. control and this social predictor was evident.

Table III–21 does show some evidence of interaction although of borderline statistical significance. The index here is the depression measure derived from the adjective checklist. It is evident that cases who subsequently died were higher on depression (one-half of a standard deviation) at the initial interview; all the other three groups were otherwise quite comparable. This provides mild support for the idea that the combination of psychological vulnerability (depressed state) and stress (relocation experience) is needed to reveal adverse health effects.

Much can be done to pursue the suggestive leads in table III–21. Interviewer's ratings of sadness correlate $r=0.52$ with self-reported depression; however, the two variables present a somewhat different pattern of findings, the reasons for which we do not understand at present. Other indices of social isolation can be examined and perhaps an overall index constructed. The interplay between social isolation and affective states (such as depression) should also be investigated.

Table III–22 shows the further exploration of the role of psychosocial predictors of health outcomes. The pattern of analysis involves selecting a variable indicative of heightened vulnerability of the respondent and seeing whether it contributes to adverse health outcomes, either by itself or in interaction with stress (i.e., case vs. control classification). The data on stroke reveal a higher incidence of new disease among respondents initially rated as moderately or severely depressed. Significance testing, following the method of Snedecor and Cochran (*38*) for analysis of proportions in two-way tables, reveals the two main effects to be reliable, but no interaction effect is evident.

The data on angina pectoris are analyzed in relation to financial assistance and gifts from family members, mostly offspring. We interpret this variable as an indicator of social support, a class of variables which is receiving increasing attention in studies of social factors in health (*39*, *40*). It is evident that both main effects and the interaction are significant. (However, because of the fact that there were no respondents who developed angina among those with some assistance, the test for interaction, in effect, becomes a test of the difference between cases and controls among respondents with no assistance.) We interpret these results as encouragement for the further exploration of social support and its effects on health outcomes.

Table III-21.
Some predictors of mortality

Interviewer's rating at initial interview	Number of cases and controls	Percent deceased
Happy	199	8.0
Serious, subdued	58	10.3
Somewhat unhappy	108	19.4
Sad		
Close to tears		
Total	365	11.8

Gamma = 0.37; $p < 0.005$.

Number of neighbors respondent knows well enough to call on (at initial interview)	Number of cases and controls	Percent deceased
None, one	170	15.9
Two or more	184	9.2
Total	354	12.4

Fisher exact test $p = 0.04$.

	Mean depression (in standard scores) at initial interview	
Cases:		
Survived	−0.01	Interaction $p < 0.07$
Deceased	.50	
Controls:		
Survived	−.06	
Deceased	−.03	

Table III-22 also shows the results of the examination of the incidence of intermittent claudication in relation to a commonly used (but temporally distant) indicator of vulnerability, growing up in an intact family vs. a home in which one or both parents were absent

Table III–22.

Some predictors of incidence of new disease

STROKE

Interviewer's rating at initial interview	Cases vs. controls	N	Percent with stroke	Significance of effects	
Happy, serious	Cases	69	14.5	Case vs. control	$p < 0.05$
	Controls	60	5.0	Int. rating	$p < 0.05$
Unhappy, sad, close to tears	Cases	49	20.4	Interaction	n.s.
	Controls	46	15.2		

ANGINA PECTORIS

Assistance and gifts from family members (initial interview)	Cases vs. controls	N	Percent with A.P.	Significance of effects	
None	Cases	97	15.5	Case vs. control	$p < 0.02$
	Controls	85	5.9	Fin. assistance	$p < 0.001$
Some	Cases	19	0.0	Interaction	$p < 0.02$
	Controls	11	0.0		

INTERMITTENT CLAUDICATION

Respondent's parents both alive and present when R growing up	Cases vs. controls	N	Percent with I.C.	Significance of effects	
Yes	Cases	89	7.9	Case vs. control	n.s.
	Controls	61	11.5	Parents present	n.s.
No	Cases	39	20.5	Interaction	$p < 0.02$
	Controls	36	5.6		

because of death or dissolution of marriage. The results show no main effects, but a significant interaction. Since this psychosocial variable, growing up in a broken home, appears to be a risk factor for higher depression and poor mental health in adult years (*41*, *42*), the similarity to the interactive effect of self-reported depression and case-control status on mortality (table III–21) is noteworthy.

From the above sample of findings it is clear that a systematic exploration of the effects of the various psychosocial factors on adverse health outcomes will be a long and complex undertaking. Moreover, this complexity is not simply a statistical, data analysis issue; it is a conceptual-theoretical complexity as well. One illustration will serve to make this point. Many of the variables collected at initial interview regarding friends and neighbors (see table III–19) may be viewed as indicators of psychosocial vulnerability; that is, elderly who have few friends, know few neighbors, and get together with them infrequently, may be viewed as social isolates who do not have much social support available to them as a buffer in difficult, stressful times. Such individuals should be at greater risk for poor health outcomes. On the other hand, several studies, particularly Fried's (*33*), suggest that "grieving for a lost home" will be particularly severe among those who had a strong pre-relocation commitment to the neighborhood, who had a greater number of close friends in the area, and who had positive feelings about their neighbors. On the basis of these results, one might predict that the social isolates will experience fewer adverse health effects, since the relocation represents little if any social uprooting to them. If both hypotheses are plausible and do, in fact, represent on going processes, one may well find no differences since the two effects will cancel each other out. Consequently, we need more probing—more subtle measures which would enable us to detect separately the presence of both processes.

In light of the above speculation, the following preliminary results are suggestive. Respondents were classified as "social isolates" if they answered "never" to both of the following questions at initial interview: "How often do your neighbors come to visit you here?" and "How often do you go to visit them in their homes?" Incidence of new cases of angina pectoris among cases who were social isolates was 22.2 percent, while incidence among the remainder of cases was 8.2 percent; the incidence among controls was 4.4 percent, irrespective of amount of contact with neighbors ($p = 0.05$, based on a chi-square analysis of trend). However, if one classifies the cases on the basis of somewhat different, but highly correlated variable, the results are different: cases who report none or only one close friend in the neighborhood at initial interview have an incidence of A.P. of 11.3 percent, while those with two or more close friends have an incidence of 12.0 percent. The fact that two highly correlated variables show differential results consistent with the two different hypotheses mentioned above suggests that it may be possible to construct a derived index which would be sensitive to these differential hypotheses.

Summary

Overall, the present study deals with a common and significant experience of social change in the lives of poor elderly residents of urban communities: The residential move from an old, frequently deteriorating neighborhood to federally subsidized elderly housing, located somewhere else in the community. For the elderly residents who make this move with the assistance of a relocation agency, this relocation may represent a desired change which involves better housing and a better opportunity for valued social activities. At the same time, the relocation may also represent a social uprooting experience for which the elderly was not quite prepared, particularly if external circumstances made it impossible to remain at the old residence.

The study involved 225 elderly (over 61) who moved (relocatees or cases) and 175 matched neighborhood controls who had not moved. Data were collected on four occasions, the last being a 2-year followup. Structured home interviews and modified medical examinations were the basis for most of the data collected: Health, psychological state, physiological data, and social behavior.

The major findings, based on a preliminary analysis of a variety of measures, may be summarized as follows: (1) Perceptions of the neighborhood and of the dwelling unit showed that the cases experienced the move as a definite improvement in the residential environment; (2) Data on friends and neighbors revealed that cases experienced some reduction in social contact with friends; however, subsequent to the move, they were more likely to indicate that their closest friend ("confidant") lived in the same building, and in general, they felt they knew their neighbors better than in the old neighborhood; (3) No significant differences in 2-year mortality rates between cases and controls were observed; (4) On a number of diverse indicators there was evidence that cases had experienced more adverse health status outcomes: nursing home admissions, hospitalizations, operations, new cases of stroke and angina pectoris, doctor's visits, and global self-rating of health status; (5) Additional analyses of selected psychosocial variables, presumed to be indicative of increased vulnerability (e.g., depression, social isolation, and low social support), revealed associations with mortality and morbidity.

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Discussion

Hogue: I would like to ask about your "number of neighbors known" measure. Would it not actually be more reasonable from a conceptual point to divide that measure into zero or none and some, the way you did your contact with neighbors?

Kasl: In fact, we originally split it on zero/one plus. I suggested splitting it at the median because the statistical test is more powerful with an even split. I think the answer is that with zero, the "n" is even smaller and it would be even rougher to handle the data analysis to get anything. Conceptually, you may be right. One would like a median split for some reasons and the zero split for other reasons.

Gruenberg: When you took your controls from nearby areas was urban renewal going on? Did the controls experience a change in neighborhood?

Kasl: Yes, but it was not really urban renewal; it was more like condemnation of single buildings. The change was sort of a piece by piece destruction, not urban renewal.

Gruenberg: Because of these buildings being destroyed, did the neighborhood get smaller in number?

Kasl: We had about five controls who did move and so they were put together with the cases. Otherwise, the basic dilemma was this: If you find truly comparable controls, they will be living in a similar neighborhood and housing, and thus are not in a very stable residential situation. They, too, may have to move soon, or the neighborhood around them is changing. On the other hand, if controls come from a stable residential setting, then—compared to cases—they are more likely to live in adequate housing, to be somewhat better off financially, to own their own house, to be married, and so on. This was not an ideal neighborhood for studying the steady state among the controls.

Gruenberg: I just wanted to know whether you had any picture of the controls going to a changed environment because their neighbors had been taken away from them. You know, people are depending more and more on living in a changing world; or on sitting still because the world around them is changing. I just wondered how much the control's world is being changed.

Kasl: Well, the only thing I can say, as shown in table III-18, is that we were seeing some changes in the cases and the controls as well. For example, 1 year later, more controls were describing their (same) neighborhood as "safe." But this is probably more a change in their standards of comparison. What you are probably asking is more subtle and I doubt whether we have measured them.

Ostfeld: The study was conducted at the tail end of the relocation process in three communities and our study subjects were substantially the last batch of relocatees. The changes in the New Haven, Hartford, and West Haven neighborhoods were probably about 80 percent concluded by the time we got into this. I would say that for the comparison group, the neighborhoods had changed over the past few years, but that they were just beginning to be stable.

Cohen: I think this gives us an opportunity we do not ordinarily have, that is, to separate out the kind of major life change comparison in the literature and the ongoing assessment of the kinds of hassles in the everyday social and psychological environment. To some people the latter are much more important and much harder to cope with than the major life events where extra resources can be managed.

Ostfeld: The event without the perception of it is a meaningless measure.

Kasl: I find that studying one "life event," like job loss or relocation, is already so complicated that I do not see any chance that the usual methodology in life change studies, the use of the schedule of recent experience which lumps many life events, can be sensitive to these complexities.

Adelman: I note that you chose some physiological parameters which are notorious for changing in response to some kind of stress or challenge, rather than their resting values as a function of increasing age. Have you considered measuring this in a slightly different way. For example, the ability to handle a load of glucose as in a glucose tolerance test? Or the response of serum cholesterol to starvation? Or blood pressure to exercise? These measures would perhaps give you a much better parameter of this type of event.

Kasl: These parameters were picked with the hope that they might be sensitive to stress. It was difficult enough to get blood from them, let alone get them to agree not to eat. You know, in a field setting it is hard to do.

Adelman: I recognize that, but I think in terms of people who perform your type of measurements, we have to recognize that, from a biological point of view, it is the acute response to a challenge that is most susceptible to age difference.

Ostfeld: These are about one-fourth of the blood chemical measurements that we made.

Kasl: We have those which the 10-channel autoanalyzer provides. No, I had become very disenchanted with measuring blood pressure at one point and then again, 2 months later hoping that it would reflect the intervening stress. I found in some other data that uric acid might be a better indicator for such a purpose. But blood pressure measurements (and these are generally averages of two values for the interview so they are a little more stable) may not be up to the task unless the monitoring is much more frequent.

Adelman: I did not make myself clear. What I am talking about is within a given group of people at a particular time, the response to a challenge over a short period of time—like a Goods-Collins test over several hours. One group versus another might be quite different, whereas you would not find a response in the resting blood sugar.

Hatano: Do you have any information on the gap between incidence and mortality? The incidence rate of stroke and angina pectoris seems to be higher among the cases and mortality seems to be higher among controls.

Ostfeld: The simplest explanation I can give is that the data Dr. Kasl presented are for those people who gave four complete interviews and four sets of blood data. We are not presenting data on those who missed the second interview. About 30 percent of them missed the second interview and then came back for the third or fourth. If we presented the data for the whole group, conceivably it could be different. I would rather not speculate beyond that. We need a more detailed analysis.

Kasl: The mortality data include everybody who started the study. Since the participation rate of the controls is somewhat lower to start with, I think they were more willing to stick with a schedule. Thus, in this complete data set, there are more controls and fewer cases.

Palmore: I want to ask you about a problem dealing with data analysis of this type. You may have run 50, 100, 150 different analyses of variance or correlations. When you run 100, you expect five of them to come out significantly different at the 0.05 level, just by chance. So, how do you handle this problem?

Kasl: I think some of the data analysis was specifically oriented toward taking only a limited set of predictors, all of which might be interpreted as indicators of greater vulnerability. Now, this still involved a large number of runs. There is no good way to do the analysis on only half of the subjects and cross-validate on the other half, since the numbers would get so small. So, ultimately, it will be the sense of the overall pattern of findings and whether they hang together or not.

G. Brody: From the predictors and the numerous interactors examined, I found that all the significant factors went in the direction of the previously stated hypotheses. That is really the best evidence I can offer that we are not seeing random change here.

Palmore: I guess my feeling is that any kind of multipurpose study like this cannot really be precise enough to avoid that problem and that you just have to admit that this needs to be replicated before you really believe it.

Gruenberg: I wonder if you might tell us a little bit more of what was being rated by the interviewer as happy, serious, not happy, in the rating of affective status. What were the interviewers reacting to?

Kasl: There was another category under the "not happy" which included "crying," so part of it was behavioral. Of course, the interviewers were aware of what the subject was saying about himself or herself in the interview. I am not sure what else they were paying attention to. Do you have any idea?

Ostfeld: This is not answering the question, but the one thing that we can look at is the fact that the blood pressure seemed to vary with the rating of mood. That is, the people who were perceived as weakened or who were perceived as having a sad affect had higher pressures.

Palmore: Let me clarify that. Was it only the interviewer's rating that predicted this? I presume you got a self-rating and that did not predict.

Kasl: Not as strongly, but the two are correlated and both are going in the same direction. In my opinion, the interviewer's rating is less likely to be involved with the correlation with health status than is the self-rating.

Schneider: Just a naive question, does bad mood reflect health status? Is a happy person a well person?

Kasl: It may be a consequence. Traditionally, studies of elderly subjects show moderately strong associations between well-being or mood and self-assessed health status. In the present study, the interviewer's rating was not associated with age, monthly income, or prior history of cardiovascular disease, which were the three predictors of mortality in our subjects.

Functional Abilities of the Elderly: An Update on the Massachusetts Health Care Panel Study*

Laurence G. Branch, Ph. D.

Department of Preventive and Social Medicine
Harvard Medical School
25 Shattuck Street
Boston, MA 02115

The challenge of providing needed services for older people from the most efficient and beneficial setting is common to both Federal and State planners. Meeting this challenge of providing services in a manner efficient for the provider and beneficial for the recipients requires that many issues be addressed simultaneously; and, if not settled, at least temporarily resolved. The periodic reconsideration of an issue as fundamental as the basic definition of aging contributes to the creation of a climate conducive to meeting this challenge. Furthermore, reviewing broader issues such as the biological, social, or psychological correlates of aging, as we have been asked to do at this conference, is a necessary antecedent to solving the problems facing older people. And just as it is beneficial to reconsider basic issues from time to time, it is also helpful to reacquaint ourselves with the patterns of behaviors, practices, and needs as older people themselves report. Such self-reported information has supplied the quantitative base for the analyses presented in this paper.

The purpose of these analyses is to examine the influences upon three kinds of functional abilities:

(1) The final functional disability of death;
(2) The functional abilities required to live independently or at least without extensive formal social service support systems; and
(3) The functional abilities necessary to accomplish the traditional activities of daily living.

The examination of factors which influence these functional abilities emphasizes the biologic correlates as much as possible, though the interrelationships among biologic, social, and psychologic factors certainly cannot be minimized.

Methods

A statewide probability sample of 1,625 noninstitutionalized elderly and 386 chronically disabled people aged 18 to 65 were interviewed from November 1974 through February 1975 (time 1), as part of the Massachusetts Health Care Panel Study. The purposes of this original study were to determine: (1) The need for long-term care facilities; (2) The health status and health service needs; and (3) The patterns of unmet needs for other nonhealth services for these high risk target populations. The areas of focus in the 45-minute personal interviews included: perceived level of functioning, activities of daily living, health care history, services currently received, morale, family and social environment, housekeeping, food shopping and preparation, transportation, emergency assistance, housing and personal economics, In short, the questionnaire assessed the level of basic or raw needs of the individuals: How they went about meeting those needs; and what parts, if any, of the basic or raw needs were as yet unmet.

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The Administration on Aging, HEW, funded a followup survey (time 2) to assess the short-term stability (15 months between interviews) of the needs of the elderly in this Massachusetts Health Care Panel Study. Of the 1,625 original elderly respondents, 1,317 were reinterviewed, 146 were not eligible respondents (103 had died, 26 were institutionalized, 17 had moved out of the State), and 162 were not able to be reinterviewed for usual reasons (temporarily absent, illness, refusals).

Results

Death—the final functional disability.—An investigation of data from two points in time was undertaken to ascertain whether any of the information provided at time 1 could discriminate between those who had died during the interim (N=103) and those who were alive and interviewed (N=1,317). The first step was to determine which specific items the deceased had answered differently. The second step was to see if these items or some subset of them could be combined by means of multiple regression analysis to produce an equation which predicts on the basis of self-reported information who is likely to die during an ensuring 15 months. The third step was to determine the individual predictive capability of some of the items identified by the multiple regression analysis.

Forty-six of the 169 variables or items from the first interview were answered significantly differently by those who were to die in the short term compared to those who did not. A summary of this phase of the analysis has been presented previously (*1*). Most of the items which did discriminate between the two groups were expected. These included age, sex, perceived health, Rosow's functional health scale (*2*), using aids to walk, number of hospitalizations in previous year, and receiving assistance for the traditional activities of daily living. Some of the other items which also discriminated might not have been expected. These items included regularly receiving B–12 injections by those who would die, frequency of getting out of the house or talking on the telephone, and interviewer judgment of orientation. Some of the items which did *not* discriminate might be surprising by their absence. These included frequency or location of physician visits, who helps them with their traditional activities of daily living or how they are assisted, type of health care coverage, morale as frequently measured (*3*), marital status, having someone they feel particularly close to, pattern of usual night's sleep, type of housing and housing satisfaction.

The next step of the analysis was to determine how these variables which discriminated at the 0.001 level would combine in a regression equation. The regression analysis included only four variables which increased R-squared by 1 percent or more. In more practical terms, this means that the regression analysis included only those four elements which had more than a trivial influence on explaining the outcome viarable of whether the person would die or not.

The four vairables which defined an older person who had a greater likelihood of dying within the short term of 15 months were reporting that one

(1) is no longer self-sufficient in personal care, like brushing hair, shaving or cutting toenails;
(2) is no longer able to do heavy work around the house, like shoveling snow or washing windows, walls or floors without help;
(3) is male; and
(4) is unable to undergo a 45-minute interview for any one of several reasons.

The next question is to quantify more exactly what the likelihood of dying is, depending on how individuals answer two simple questions and their gender (table III–23). The procedure for authorizing a proxy interview in this type of survey research is very complicated, and consequently cannot be applied easily to other planners or clinicians. Therefore, this variable is omitted from the following subgroup analyses. Overall, about 1 in 15 or 7 percent of our sample of older people died during the 15 months following the first interview. If an older person reports that (s)he is no longer self-sufficient in personal care (which 13 percent of the sample reported), then the clinician and/or planner knows that this person's likelihood of mortality during the short term is increased to about one in five.

The chances are about 2 in 15 that a person who reports no longer being able to do heavy work around the house (which 43 percent so reported) will die in 15 months. Males were 40 percent of the sample and their chances of mortality during 15 months were about 1 in 10, independent of how they answered any other questions in the interview.

If a person reports both that (s)he is no longer self-sufficient in personal care and no longer able to do heavy work around the house (as 11 percent did), then the likelihood of mortality within 15 months is about one in four (24 percent). If a male makes those responses, the likelihood becomes two out of five (40 percent). If female, the likelihood of short-term mortality is about one out of six (17 percent).

The only other response pattern which indicated a substantially greater than average likelihood of short term mortality was for males to report that they are no longer able to do heavy work around the

Table III–23.
Likelihood of mortality within 15 months for certain groups of older people

Response patterns of older people	Likelihood of response pattern occurrence (percent)	Of these, likelihood of mortality within 15 months
Total probability sample	100	7 percent (about 1 in 15)
Single items:		
Not self-sufficient in personal care	13	21 percent (about 1 in 5)
No longer able to do heavy work around the house, like washing walls, shoveling snow.	43	13 percent (about 2 in 15)
Male	40	11 percent (about 1 in 10)
Selected combinations:		
Not self-sufficient in personal care AND No longer able to do heavy work around the house like washing walls, shoveling snow	11	24 percent (about 1 in 4)
Not self-sufficient in personal care AND No longer able to do heavy work around the house like washing walls, shoveling snow. AND Male	3	40 percent (about 2 in 5)
Not self-sufficient in personal care AND No longer able to do heavy work around the house like washing walls, shoveling snow. AND Female	7	17 percent (about 1 in 6)
Self-sufficient in personal care AND No longer able to do heavy work around the house like washing walls, shoveling snow. AND Male	13	16 percent (about 1 in 6)

house, even though self-sufficient in personal care. Thirteen percent of the sample were in this subgroup, and about one out of six of these died within the 15 months.

Having identified the specific subgroups among older people with increased likelihood of mortality in the short term, planners and clinicians might also want to know whether any response patterns to these two simple questions are indicative of a substantially decreased likelihood of short term mortality. Independent of gender, those reporting that they are able to do the heavy work around the house (which 57 percent so reported) had only 3 percent mortality in the short term, compared to the total sample rate of 7 percent.

Functional abilities required for complete independence.—The remaining analyses concentrate on understanding the factors which influence certain functional abilities for the living, specifically those older people interviewed at time 2. One particular set of functional abilities, one which has serious implications for the type and amount of public support services which an older person might require, is the individual's ability to accomplish certain activities normally required to live independently in the community or at least outside of an institution. The assessments used to gauge an older person's needs for formal assistance were based on both self-reported behavioral information and self-reported evaluative information. The need assessment areas are the ones for which the public support system is often relied upon to provide either home-based or institutionally based services, such as transportation, personal care, housekeeping, social activities, emergency assistance, food shopping, and food preparation. The specific operational definitions employed are critically important in a need assessment study, and consequently the exact definitions are presented in appendix A.

Each of the definitions differentiated the respondents into four major categories:

(1) Need currently met and no apparent problem.
(2) Need currently met but potential problems are apparent.
(3) Uncertain whether need currently met and potential problems are apparent.
(4) Need currently unmet and current problems apparent.

Each of these major categories in turn contained several subcategories which ordered the various response profiles that occurred from greater self-sufficiency to lesser self-sufficiency.

The need assessment definitions generally considered the behavioral component more salient in the determination of need than the evaluative component, but the evaluative component was more important in judging whether a potential problem might became manifest. Neither the behavioral nor the evaluative component, alone, is sufficient for categorization at the extremes.

For the social activities need assessment, the behavioral and the evaluative components were each reduced to a single numerical index by combining the responses from each of the items to form an additive index. The scoring of each index was simple enough; a response suggesting the best possible alternative was scored as one, the worst possible alternative was scored as five, and the neutral point or a missing answer was scored as three. The behavioral component of the need assessment for housekeeping assistance was also an additive scale with four points, with the responses between the neutral and the worst possible points scored as four.

Table III–24 presents a summary of the need assessment findings. The percentage of noninstitutionalized older people at time 2 who were *unable* to take care of their needs under their present arrangements were as follows: 7 percent in transportation; 3 percent in personal care; 2 percent in housekeeping; 2 percent in social activities; 2 percent in emergency assistance; 1 percent in food shopping; and ½ percent in food preparation. The vast majority were fully independent and self-sufficient in their present situations. About 9 out of 10 older people had no unmet social service needs, and only 4 percent had two or more unmet social service needs.

The response distributions for each of these need assessment areas were subjected to subgroup analyses designed to determine whether any association existed between the subgroups variables—age, gender, household composition and perceived health—and the particular need area's response distribution over the four major categories. Table III–25 presents the results of these subgroup analyses. Though the information obtained for each need assessment area has many implications which are discussed elsewhere (*4*), the focus at present is to summarize the overall influences of the subgroup variables on the need assessments as a group.

The biologic variable of age was associated with significant differences in need assessment categorizations in four areas—transportation, personal care, social activities, and food shopping. The associations found in the transportation need assessment most closely paralleled the associations found for much of the self-reported information—those aged 85 or over clearly reported the most negative patterns and situations; those aged 80 through 84 also reported negative patterns and situations, but to a slightly lesser degree than those aged 85 or over; those under age 80 reported favorable patterns and situations for the most part. A second type of association was noted between age and two of the need assessment variables—social activities and food shopping—namely a straightforward linear relationship in which each increase in age was associated with a steady increment in the more negative response patterns. It should be emphasized that this linear relationship did not occur very often across all the other self-reported variables, but nevertheless did occur with these two assessment variables. A third type of relationship was noted between age and personal care, but noted only once in all the other self-reported variables. The age relationship with personal care was that those aged 80 through 84 reported the most serious problems, while those aged 85 or more re-

Table III–24.

Summary in percentages of social service needs assessment for the elderly at time 2

		1. Need met, no apparent problem				2. Need met, potential problem				3. Uncertain need met, potential problem	4. Need unmet, current problem
	Number	Total	Self sufficient	Within house support system	Outside house support system	Total	Self sufficient	Within house support system	Outside house support system		
Transportation	(1, 290)	69	51	11	7	20	5	7	8	4	7
Personal care	(1, 282)	85	—	—	—	12	—	—	—	—	3
Housekeeping	(1, 289)	86	50	29	7	3	3	1	1	9	2
Social activities	(1, 314)	70	—	—	—	23	—	—	—	4	2
Emergency assistance	(1, 293)	78	—	—	—	9	—	—	—	11	2
Food shopping	(1, 302)	86	59	21	6	9	2	5	2	4	1
Food preparation	(1, 286)	86	54	30	2	10	7	2	⅓	3	⅓

Table III–25.
Statistical significance between subgroup analytic variables and various need assessments for the elderly at time 2

Need assessments	Subgroup analytic variables			
	Age	Gender	Household composition	Perceived health
Transportation	0. 001	0. 001	0. 001	0. 001
Personal care	. 001	. 01	. 001	. 001
Housekeeping	NS	. 05	NS	. 001
Social activities	. 001	NS	. 001	. 001
Emergency assistance	NS	. 001	. 001	. 05
Food shopping	. 001	NS	NS	. 001
Food preparation	NS	. 01	. 001	. 001

ported more favorable patterns. It seems those who had been in need had ultimately come to rely on other people for help in this area. Apparently the problems in personal care begin around age 80 as they did for other things, but the problems are solved by age 85 rather than deteriorating more seriously.

Gender was associated with significant differences in five need assessment areas—transportation, personal care, housekeeping, emergency assistance, and food preparation. In every instance females had higher levels of unmet needs or greater problems than males, including the gender-stereotyped areas of housekeeping and food preparation. Several explanations for this are possible. One is that the increased longevity of women over men might possibly be due in part to an increased number of women who are surviving with unmet needs in any one of several social service areas. In other words, perhaps the men with unmet needs are not surviving to the degree that women with unmet needs are.

Household composition was associated with significant differences in five areas—transportation, personal care, social activities, emergency assistance, and food preparation. The household composition was consistently associated with more problems or the more negative response pattern was living with children only. Those living with a spouse only often reported the most favorable patterns and situations. The definition of an unmet need in emergency assistance presupposed that the older person was living alone, so that particular household composition was associated with the most negative pattern in emergency assistance.

The respondents' perception of their own health was also associated with significant differences in every one of the need assessment areas and the pattern was remarkably consistent. Those reporting excellent, good or fair perceived health were very similar in response patterns; those reporting poor perceived health were dramatically different from the rest of the elderly. In these need assessment areas, those reporting poor health actually had a fourfold to fivefold increase in the percentage with an unmet need compared to the rest of the elderly.

Functional abilities necessary for the traditional activities of daily living.—The basic activities of bathing, dressing, toileting, transferring, continence, and eating have demonstrated research and evaluation utility (*5*, *6*). Many people have limited abilities in some of these areas following serious accident or illness episodes. The elderly respondents of this panel study reported their capabilities in a modified activities of daily living (ADL) index, modified because the activities of toileting and continence were excluded from the self-reported interviews while the activities of personal grooming and walking across a small room were included.

Table III–26 presents a summary, for the two points in time and during the 15-month interim, of the percentage of older people who reported independence or relying on another person to accomplish at least some part of the activity. Several aspects of the information are noteworthy.

First, notice that the response patterns are very similar at the aggregate level for the two snapshots in time, and in fact, the two snapshots present patterns which are within 2 percentage points of each other. Admittedly, items like these which have the vast majority of respondents reporting the same thing (independence) do not highlight differences over time. Nevertheless, the general pattern found for nearly every item including those with more even distributions had very little fluctuation over the short term of 15 months.

Second, since there was such constancy at the aggregate level over time, the most serious remaining question for planners is to gauge the turnover rate among service recipients. That is, if one knows that there will be about a thousand older people in a state needing a formal support service at any point in time, it then becomes important to know whether the thousand at any point in time come from a larger group of 2,000 or of 10,000. If 2,000, the implication is that the people in need of formal support services tend to have that need for a much longer duration than if the group was 10,000, which implies that the rate of a particular need fluctuates markedly among the larger group.

The interview schedule elicited responses about the practices and events which occurred between the interviews whenever appropriate as a means of gauging the size of the group from which those in need at a point in time are drawn.

The information shown in table III–26 about the percentage of people relying on other people to accomplish those activities of daily living during the interim suggests that the group is almost twice as large as the number of older people who required assistance at either point in time. The rate of twice as many or less reporting a problem during the interim compared to the rate of reporting the same problem at a specific point in time was found in many other instances as well, specifically those receiving special service such as speech or rehabilita-

Table III–26.
Activities of daily living for the elderly at two points in time and during the interim, in percentages

Modified activities of daily living	Time 1			Interim			Time 2		
	Independent	Other people involved	Percent feeling need for additional help	Independent	Other people involved	Percent feeling need for additional help	Independent	Other people involved	Percent feeling need for additional help
Bathing	94	6	3	89	11	2	92	8	2
Dressing	96	4	2	93	7	1	96	4	1
Transferring	97	3	([1])	95	5	([1])	98	2	([1])
Eating	99	1	1	97	3	1	99	1	½
Grooming	89	11	2	90	10	2	91	9	1
Walking across a small room	99	1	5	91	9	3	97	3	2

[1] Not ascertained.

tion therapies, professional counseling, home-based nursing services or hot meal services and injections.

Table III–26 also presents the percentage of people who feel they could use help beyond what they are receiving for specific activities. In most surveys the assumption is made that those receiving assistance have solved their problems and the research objective is merely to quantify the percentage not receiving assistance who should. As the data in table III–26 indicate, about one out of every four or five older people who was receiving help from another person in the activities of daily living felt that (s)he actually needed more help than (s)he received.

Implications for Future Research

Not surprisingly, the older people in this study reported very different response patterns for the three types of functional abilities reviewed here. It is true that the analyses of the response patterns led to many generalizations about the factors influencing such response patterns. Nevertheless, the very decision to review three types of functional abilities highlights the fact that the concept of "functional abilities" is amorphous and to a considerable extent the functional abilities of the elderly will reflect those aspects which are measured in a particular study.

Some additional methodological and procedural research is required to synthesize as much as possible the various factors which are used to assess functional abilities. Perhaps some of the various approaches to measuring functional disabilities—the items presented previously which predict total disability (death); the Katz ADL index designed to discriminate functional abilities of the severely physically disabled of all ages; Rosow's functional health scale which has been very useful differentiating capabilities among the noninstitutionalized elderly (and therefore somewhat less severely disabled); individual items incorporated by Haber and by Shanas in different national surveys which discriminate functional abilities among essentially nondisabled populations; the social service need assessments presented previously which differentiate the fairly rare groups (½ to 7 percent) of older people who have specific limitations—are all focusing on different aspects of a unitary concept. Disability or functional limitations might be viewed as a continuum from low or no limitation to high or severe limitation, and each of these measurement approaches has focused on a specific part of the continuum.

A composite index of disability for the elderly would unquestionably simplify research and evaluation studies. Of course the logic underpinning this contention is the same logic which supports the need for composite health status indicators, a logic which has been stated else- (*7, 8, 9*). The applications of a composite index of disability are extremely varied.

Since the interview schedule contained three of the measures of disability (a five-item modified Katz ADL index; a four-item Rosow functional health scale; and six of the seven Social Service Need Assessment areas, with personal care omitted because the ADL index incorporated this dimension), a simplistic and preliminary test of an additive value function was made by merely summing the number of times a person was found with a disability in any of the 15 possible areas or activities. No consideration was given either to severity of limitation or to the differential importance of the activity.

The frequency distribution of the 1,317 time 2 elderly respondents on the 16-point additive index—15 areas of disability and the zero point—is presented in table III–27. Again, the overall impression is that the vast majority of the noninstitutionalized elderly have minimal functional disabilities—80 percent have 0, 1, or 2 instances of limitation—implying that the services for the elderly ought to be targeted to the few who have serious problems rather than providing minimal services for many. The data indicate that no one had a disability score greater than 10, and the 8 percent with the most serious disabilities had scores from 5 to 10.

An examination of the loadings of the individual scores on the additive index revealed that 84 percent of the index scores reflected just the Rosow score of zero to four, with zero scored for both the ADL and need assessment components. This 84 percent includes the 39 percent who had zero on all three components. For the 8 percent with scores of five or more, the ADL component contributed 141 points and the need assessment component contributed 124 points, while the Rosow component contributed 334 points. Clearly the individuals' score on the

Table III–27.
Frequency distribution of the elderly on an additive index of disability

Score	Percent	Cumulative percent
0	39	39
1	28	67
2	13	80
3	8	88
4	4	92
5	3	95
6	2	97
7	1	98
8	½	99
9	¼	99
10	¼	100
11	0	100
12	0	100
13	0	100
14	0	100
15	0	100

Rosow component is contributing most to the additive index score, with the ADL and need assessment components contributing to a much lesser extent. Though it must be borne in mind that the 8 percent with serious problems i.e., scores of five or more, by definition had disabilities beyond the four activities measured in the Rosow scale. The implications for topics of future research are immediately apparent.

Appendix A

Transportation: Operational Definitions for Need Assessment.

The following definitions are based on the response patterns reported within items 98–103. The number of cases that are included in each category is presented on the left.

Q98: About how often do you get out of your (HOUSE/APARTMENT) for any reason—almost every day, a few times a week, about once a week, several times a month, about once a month, several times a year, about once a year, never or almost never except for emergencies.

Q99: Which of these three statements best describes your present transportation pattern:

1. I am completely free to go and return as I want, when I want, and for what I want.
2. I go out for most things I need or like, but I don't do many extra things like going visiting.
3. I only go out for special occasions and/or basic necessities like food shopping.

Q100: How often are you able to go to the places you would like to—would you go as often as you'd like, most of the time, or not nearly as often as you'd like?

Q101: When you are going someplace that is too far to walk how do you usually get there—by car, public transportation, taxi, or what?

Q102: When you go somewhere by car, who usually drives—do you usually drive yourself, does someone living here drive you, or does someone else usually drive you? (Who is that?)

Q103: Do you find getting where you need to go is usually a big problem, a little problem, or no problem at all?

Number		**Categories**
905		1. **Need Met, No Apparent Problem**
	789	1.1 Transportation pattern—completely free (Q99); and no problem with transportation (Q103).
	65	1.2 Transportation pattern—completely free (Q99); and a little problem with transportation (Q103).
	19	1.3 Transportation pattern—goes out for most things (Q99); no problem with transportation (Q103); usual mode of transportation is a car (Q101); and usually drives self (Q102).
	17	1.4 Transportation pattern—goes out for most things (Q99); no problem with transportation (Q103), and usual mode of transportation is a taxi or public transportation (Q101).
	5	1.5 Transportation pattern—goes out for most things (Q99); a little problem with transportation (Q103);

			usual mode of transportation is a car (Q101); and usually drives self (Q102).
	10	1.6	Transportation pattern—goes out for most things (Q99); a little problem with transportation (Q103); and usual mode of transportation is a taxi or public transportation (Q101).
245		2.	**Need Met, Potential Problem**
	8	2.1	Transportation pattern—completely free (Q99); and a big problem with transportation (Q103).
	19	2.2	Transportation pattern—goes out for most things (Q99); no problem with transportation (Q103); usual mode of transportation is a car (Q101); and a household member usually drives (Q102).
	19	2.3	Transportation pattern—goes out for most things (Q99); no problem with transportation (Q103); usual mode of transportation is a car (Q101); and a person outside of household usually drives (Q102).
	11	2.4	Transportation pattern—goes out for most things (Q99); a little problem with transportation (Q103); usual mode of transportation is a car (Q101); and a household member usually drives (Q102).
	18	2.5	Transportation pattern—goes out for most things (Q99); a little problem with transportation (Q103); usual mode of transportation is a car (Q101); and a person outside of household usually drives (Q102).
	13	2.6	Transportation pattern—goes out for most things (Q99) and *Either* a big problem with transportation (Q103) *Or* able to go places not nearly as often as would like (Q100) and not answered if there is a problem with transportation (Q103).
	16	2.7	Transportation pattern—goes out for basic necessities (Q99); no problem with transportation (Q103); usual mode of transportation is a car (Q101); and usually drives self (Q102).
	26	2.8	Transportation pattern—goes out for basic necessities (Q99); no problem with transportation (Q103); and usual mode of transportation is a taxi or public transportation (Q101).
	50	2.9	Transportation pattern—goes out for basic necessities (Q99); no problem with transportation (Q103); usual mode of transportation is a car (Q101); and a household member usually drives (Q102).
	49	2.10	Transportation pattern—goes out for basic necessities (Q99); no problem with transportation (Q103); usual mode of transportation is a car (Q101); and a person outside of household usually drives (Q102).
	16	2.11	Transportation pattern—goes out for basic necessities (Q99); a little problem with transportation (Q103); and able to go places as often or most of the time as would like (Q100).
54		3.	**Uncertain Need Met, Potential Problem**
	5	3.1	Transportation problem—goes out for basic necessities (Q99); a little problem with transportation (Q103); able to go places not nearly as often as would like (Q100); usual mode of transportation is a car (Q100); and usually drives self (Q102).

	9	3.2	Transportation pattern—goes out for basic necessities (Q99); a little problem with transportation (Q103); able to go places not nearly as often as would like (Q100); usual mode of transportation is a taxi or public transportation (Q101).
	9	3.3	Transportation pattern—goes out for basic necessities (Q99); a little problem with transportation (Q103); able to go places not nearly as often as would like (Q100); usual mode of transportation is a car (Q101); and a household member usually drives (Q102).
	23	3.4	Transportation pattern—goes out for basic necessities (Q99); a little problem with transportation (Q103); able to go places not nearly as often as would like (Q100); usual mode of transportation is a car (Q101); and a person outside of household usually drives (Q102).
	3	3.5	Transportation pattern—goes out for basic necessities (Q99); a little problem with transportation (Q103); and missing data on how often able to go places would like (Q100).
	5	3.6	Transportation pattern—goes out for basic necessities (Q99); a big problem with transportation (Q103); and able to go places as often or most of the time as would like (Q100).
93			**4. Need Unmet, Current Problem**
	2	4.1	Transportation pattern—goes out for basic necessities (Q99); a big problem with transportation (Q103); able to go places not nearly as often as would like (Q100); usual mode of transportation is a car (Q101); and usually drives self (Q102).
	18	4.2	Transportation pattern—goes out for basic necessities (Q99); a big problem with transportation (Q103); able to go places not nearly as often as would like (Q100); usual mode of transportation is a taxi or public transportation (Q101).
	10	4.3	Transportation pattern—goes out for basic necessities (Q99); a big problem with transportation (Q103); able to go places not nearly as often as would like (Q100); usual mode of transportation is a car (Q101); and a household member usually drives (Q102).
	25	4.4	Transportation pattern—goes out for basic necessities (Q99); a big problem with transportation (Q103); able to go places not nearly as often as would like (Q100); usual mode of transportation is a car (Q101); and a person outside of household usually drives (Q102).
	1	4.5	Transportation pattern—goes out for basic necessities (Q99); a big problem with transportation (Q103); able to go places not nearly as often as would like (Q100); and usual mode of transportation is some special service (Q101).
	7	4.6	Transportation pattern—goes out for basic necessities (Q99); a big problem with transportation (Q103); and missing data on how often able to go places would like (Q100).
	30	4.7	(HOMEBOUND) goes out of home never or almost never except for emergencies (Q98).
18			**Omitted Due to Missing Data**

2 **Omitted Due to Other Pattern**

Combined Personal Care Based on Five Activities: Operational Definitions for Need Assessment.

The following definitions are based on the response patterns reported within items 41–45, 50–51, 53–57, 59–63, 65–70, 72–73. The number of cases that is included in each category is presented on the left.

Q41: I need to find out about some activities of daily living. I know you've given me some of the information already, but I need to make sure I get it down correctly in this section.

Since the last interview, was there any period in which another person generally helped you to walk across a small room?

Q42: Who usually helped? (Get Relationship)

Q43: Are you still getting this help?

Q44: Since the last interview, was there any time you felt you needed some (extra) help getting around the house, but didn't have anyone to help you on a regular basis?

Q45: Do you still feel you could use some (extra) help at the present time?

Q50: Since the last interview, was there any period in which another person generally helped you to get dressed or stayed with you while you dressed?

Q51: Who was that usually? (Get Relationship)

Q53: Are you still getting this help?

Q54: Since the last interview, was there any time you felt you needed some (extra) help to get dressed, but didn't have anyone to help you on a regular basis?

Q55: Do you still feel you could use some (extra) help at the present time?

Q56: Since the last interview, was there any period in which another person generally helped you with bathing or stayed with you while you bathed?

Q57: Who was that usually? (Get Relationship)

Q59: Are you still getting this help?

Q60: Since the last interview, was there any time you felt you needed some (extra) help with bathing, but didn't have anyone to help you on a regular basis?

Q61: Do you still feel you could use some (extra) help at the present time?

Q62: Since the last interview, was there any period in which another person generally helped you to eat or needed to be in the room with you?

Q63: Who was that usually? (Get Relationship)

Q65: Are you still getting this help?

Q66: Since the last interview, was there any time you felt you needed some (extra) help with feeding, but didn't have anyone to help you on a regular basis?

Q67: Do you still feel you could use some (extra) help at the present time?

Q68: Since the last interview, was there any period in which another person needed to be in the room with you, or generally helped you to take care of things like brushing hair, shaving, or cutting toenails?

Q69: Who was that usually? (Get Relationship)

Q70: Are you still getting this help?

Q72: Since the last interview, was there any time you felt you needed some (extra) help with your personal care, but didn't have anyone to help you on a regular basis?

Q73: Do you still feel you could use some (extra) help at the present time?

Activity No. 1 Bathing

1192 1. **No Need**
Not currently receiving help with bathing (Q56 and 59); and does not feel need for help (Q60 and 61).

93 2. **Need Met**
Currently receiving help with bathing (Q56 and 59); and does not feel need for extra help (Q60 and 61).

8 3. **Unmet and Receiving Help**
Currently receiving help with bathing (Q56 and 59); and does feel need for extra help (Q60 and 61).

13 4. **Unmet and not Receiving Help**
Not currently receiving help with bathing (Q56 and 59); and does feel need for help (Q60 and 61).

Activity No. 2 Dressing

1243 1. **No need**
Not currently receiving help getting dressed (Q50 and 53); and does not feel need for help (Q54 and 55).

56 2. **Need Met**
Currently receiving help getting dressed (Q50 and 53); and does not feel need for extra help (Q54 and 55).

2 3. **Unmet and Receiving Help**
Currently receiving help getting dressed (Q50 and 53); and does feel need for extra help (Q54 and 55).

8 4. **Unmet and not Receiving Help**
Not currently receiving help getting dressed (Q50 and 53); and does feel need for help (Q54 and 55).

Activity No. 3 Eating

1287 1. **No Need**
Not currently receiving help with eating (Q62 and 65); and does not feel need for help (Q66 and 67).

18 2. **Need Met**
Currently receiving help with eating (Q62 and 65); and does not feel need for extra help (Q66 and 67).

0 3. **Unmet and Receiving Help**
Currently receiving help with eating (Q62 and 65); and does feel need for extra help (Q66 and 67).

1 4. **Unmet and not Receiving Help**
Not currently receiving help with eating (Q62 and 65); and does feel need for help (Q66 and 67).

Activity No. 4 Grooming

1183 1. **No Need**
Not currently receiving help with grooming (Q68 and 70); and does not feel need for help (Q72 and 73).

103 2. **Need Met**
Currently receiving help with grooming (Q68 and 70); and does not feel need for help (Q72 and 73).

9	3. **Unmet and Receiving Help** Currently receiving help with grooming (Q68 and 70); and does feel need for extra help (Q72 and 73).
9	4. **Unmet and not Receiving Help** Not currently receiving help with grooming (Q68 and 70); and does feel need for help (Q72 and 73).
	Activity No. 5 Ambulation
1246	1. **No Need** Not currently receiving help walking across a small room (Q41 and 43); and does not feel need for help (Q44 and 45).
36	2. **Need Met** Currently receiving help walking across a small room (Q41 and 43); and does not feel need for extra help (Q44 and 45).
8	3. **Unmet and Receiving Help** Currently receiving help walking across a small room (Q41 and 43); and does feel need for extra help (Q44 and 45).
17	4. **Unmet and not Receiving Help** Not currently receiving help walking across a small room (Q41 and 43); and does feel need for help (Q44 and 45).

Number		**Categories**
1091		1. **Need Met, No Apparent Problem**
	1091	1.1 No need score on all five activities.
148		2. **Need Met, Potential Problem**
	85	2.1 Need met score on at least one of five activities; all other items scored as no need; and help received is from household members, but no people outside of household or paid sources involved.
	24	2.2 Need met score on at least one of five activities; all other items scored as no need; and help received is from people outside of household but paid sources not involved.
	39	2.3 Need met score on at least one of five activities; all other items scored as no need; and help received is from paid source.
—		3. **Uncertain Need Met, Potential Problem**
43		4. **Need Unmet, Current Problem**
	7	4.1 Need unmet on at least one of five activities; and help received is from household members, but no people outside of household or paid sources involved.
	7	4.2 Need unmet on at least one of five activities; and help received is from people outside of household but paid sources not involved.
	5	4.3 Need unmet on at least one of five activities; and help received is from paid source.
	24	4.4 Need unmet on at least one of five activities; and not getting any help at present.
35		**Omitted Due to Missing Data**

Housekeeping: Operational Definitions for Need Assessment.

The following definitions are based on the response patterns reported within items 74–83. The number of cases that is included in each category is presented on the left.

Q74: Who usually does most of the housekeeping like washing clothes and cleaning here? (Get Relationship)

Q75: In general ,is there any problem getting the housekeeping done, like cleaning and washing, or not?

Q76: Would you say it is a very serious problem, a somewhat serious problem, or not too serious a problem?

Q77: Does this statement pretty much describe your housekeeping situation or not: all the housekeeping gets done about the same as it did years ago; it gets done about as often, about as well, and with no more real difficulty.

Q78: Is there any problem getting the housekeeping done as often as you like?

Q80: (And) is there any problem getting it done as well as it used to be?

Q82: (And) is the housekeeping (also) a problem because it is just more difficult for you to get done?

Housekeeping problem scores based on a three item additive scale; each item scored 1, 2, 3, or 5 as indicated.

		Score
Item No. 1 Q79:	How often does the housekeeping get done now—most of the time, just some of the time, or hardly ever gets done at all?	
	no problem with how often housekeeping done (from Q77 or Q78)	5
	housekeeping gets done most of the time	3
	housekeeping gets done some of the time	2
	housekeeping gets done hardly ever	1
	missing data on how often housekeeping done	3
Item No. 2 Q81:	How well does the housekeeping get done now—pretty well, fairly well, or not at all well?	
	no problem with how well housekeeping done (from Q77 or Q80)	5
	housekeeping done pretty well	3
	housekeeping done fairly well	2
	housekeeping done not at all well	1
	missing data on how well housekeeping done	3
Item No. 3 Q83:	Does the housekeeping present a *lot* of difficulty or a *little* difficulty?	
	no problem with housekeeping being difficult (from Q77 or Q82)	5
	housekeeping presents a little difficulty	3
	housekeeping presents a lot of difficulty	1
	missing data on housekeeping being difficult	3

Number		**Categories**
1116		**1. Need Met, No Apparent Problem**
	996	1.1 Housekeeping situation the same as years ago (Q77); and no problem with housekeeping (Q75).
	43	1.2 Housekeeping problem score is 13 or more; and no problem with housekeeping (Q75).
	46	1.3 Housekeeping problem score is 10, 11 or 12; no problem with housekeeping (Q75); and housekeeping done by self (Q74).

31 1.4 Housekeeping situation not the same as years ago (Q77); no problem with housekeeping (Q75); and no specific problem with housekeeping (Q78, Q80, Q82).

44 **2. Need Met, Potential Problem**

4 2.1 Not too serious or somewhat serious problem with housekeeping (Q76); and no specific problem with housekeeping (Q78, Q80, Q82).

6 2.2 Housekeeping problem score is 13 or more; and a not too serious problem with housekeeping (Q76).

4 2.3 Housekeeping problem score is 10, 11 or 12; no problem or a not too serious problem with housekeeping (Q75 or Q76); and household member does housekeeping (Q74).

10 2.4 Housekeeping problem score is 10, 11 or 12; no problem or a not too serious problem with housekeeping (Q75 or Q76); and person outside of household does housekeeping (Q74).

12 2.5 Housekeeping problem score is 10, 11 or 12; a not too serious problem with housekeeping (Q76); and housekeeping done by self (Q74).

4 2.6 Housekeeping problem score is 13 or more; and a somewhat serious problem with housekeeping (Q76).

4 2.7 Housekeeping problem score is 10, 11 or 12; and a somewhat serious problem with housekeeping (Q76).

112 **3. Uncertain Need Met, Potential Problem**

0 3.1 Housekeeping problem score is 13 or more; and a very serious problem with housekeeping (Q76).

1 3.2 Housekeeping problem score is 10, 11 or 12; and a very serious problem with housekeeping (Q76).

48 3.3 Housekeeping problem score is 8 or 9; and no problem or a not too serious problem with housekeeping (Q75 or Q76).

10 3.4 Housekeeping problem score is 8 or 9; and a somewhat serious problem with housekeeping (Q76).

0 3.5 Housekeeping problem score is 8 or 9; and a very serious problem with housekeeping (Q76).

37 3.6 Housekeeping problem score is 6 or 7; and no problem or a not too serious problem with housekeeping (Q75 or Q76).

7 3.7 Housekeeping problem score is 7; and a somewhat serious problem with housekeeping (Q76).

9 3.8 Housekeeping problem score is 4 or 5; and no problem or a not too serious problem with housekeeping (Q75 or Q76).

26 **4. Need Unmet, Current Problem**

1 4.1 Housekeeping problem score is 6; and a somewhat serious problem with housekeeping (Q76).

3 4.2 Housekeeping problem score is 6 or 7; and a very serious problem with housekeeping (Q76).

9 4.3 Housekeeping problem score is 4 or 5; and a somewhat serious housekeeping problem (Q76).

10 4.4 Housekeeping problem score is 4 or 5; and a very serious housekeeping problem (Q76).

3 4.5 Housekeeping problem score is 3; and a somewhat or very serious housekeeping problem (Q76).

19 **Omitted Due to Missing Data**

Social Activities: Operational Definitions for Need Assessment.

The following definitions are based on the response patterns reported within items 91–98, 105, 108 and 154. The number of cases that is included in each category is presented on the left.

Social Activities behavior scores based on a seven item additive scale; each item scored 1, 3 or 5 as indicated.

		Score
Item No. 1 Q91:	About how often do you talk with friends or relatives on the telephone—several times a day, once a day, a few times a week, once a week, or less often?	
	talks with friends or relatives on telephone several times a day	5
	talks with friends or relatives on telephone once a day	5
	talks with friends or relatives on telephone a few times a week	3
	talks with friends or relatives on telephone once a week	3
	talks with friends or relatives on telephone less often	1
	missing data on frequency of talking with friends or relatives on telephone	3
Item No. 2 Q92:	About how often do you talk in person to someone who does not live with you—almost every day, a few times a week, once a week, a few times a month, once a month, or less often?	
	contact with non-household person almost every day	5
	contact with non-household person a few times a week	5
	contact with non-household person once a week	3
	contact with non-household person a few times a month	1
	contact with non-household person once a month	1
	contact with non-household person less often	1
	missing data on frequency of contact with non-household person	3
Item No. 3 Q93:	Do you generally spend most of the day with someone, or not?	
	does generally spend most of the day with someone	5
	does not generally spend most of the day with someone	1
	missing data on whether or not spends most of the day with someone	3
Item No. 4 Q95:	Is there a friend, a relative or someone you know that you feel particularly close to, that is, somebody you can be completely yourself with and in whom you have complete trust and confidence?	
	has close friend	5
	does not have close friend	1
	missing data on whether or not has close friend	3
Item No. 5 Q97:	Do you belong to any clubs, lodges or organizations?	
	belongs to a club, lodge or organization	5
	does not belong to a club, lodge or organization	1
	missing data on whether or not belongs to a club, lodge or organization	3
Item No. 6 Q98:	About how often do you get out of your (house/apartment) for any reason—almost every day, a few times a week, about once a week, several times a month, about once a month, several times a year, about once a year, never or almost never except for emergencies?	

goes out of home almost every day	5
goes out of home a few times a week	3
goes out of home about once a week	1
goes out of home several times a month	1
goes out of home about once a month	1
goes out of home several times a year	1
goes out of home about once a year	1
goes out of home never or almost never except for emergencies	1
missing data on frequency of leaving home	3

Item No. 7 Q154: Are you working at a job now?

working at a job now	5
not working at a job now	1
missing data on whether or not working at a job now	3

Social Activities evaluation scores based on a four item additive scale; each item scored 1, 3 or 5 as indicated.

Item No. 1 Q94: Would you say you see as much of your relatives as you would like, or not?

	Score
sees as much of relatives as like	5
does not see as much of relatives	1
missing data on whether person sees as much of relatives	3

Item No. 2 Q96: Do you see as much of that person as you would like, or not?

	Score
sees as much of close friend as like	5
does not see as much of close friend	1
missing data on whether person sees as much of close friend	3

Item No. 3 Q105: In general, how satisfied are you with the way you spend your time—would you say very satisfied, somewhat satisfied, or not at all satisfied?

	Score
very satisfied with how spend time	5
somewhat satisfied with how spend time	5
not at all satisfied with how spend time	1
missing data on whether person satisfied with how spend time	3

Item No. 4 Q108: How satisfied are you with your life today—would you say very satisfied, fairly satisfied, satisfied, or not satisfied?

	Score
very satisfied with life today	5
fairly satisfied with life today	5
satisfied with life today	5
not satisfied with life today	1
missing data on whether person is satisfied with life today	3

Number		Categories
920		1. **Need Met, No Apparent Problem**
	34	1.1 Behavior score 35 or 33; and evaluation score 20 or 18.
	16	1.2 Behavior score 35 or 33; and evaluation score 16.

	137	1.3	Behavior score 35, 33 or 31; and evaluation score 20, 18 or 16.
	318	1.4	Behavior score 29 or 27; and evaluation score 20, 18 or 16.
	394	1.5	Behavior score 25, 23 or 21; and evaluation score 20, 18 or 16.
	21	1.6	Behavior score 21 or more; and evaluation score 14.
306		2.	**Need Met, Potential Problem**
	123	2.1	Behavior score 21 or more; and evaluation score 12.
	3	2.2	Behavior score 21 or more; and evaluation score 10.
	49	2.3	Behavior score 19; and evaluation score 16 or more.
	6	2.4	Behavior score 19; and evaluation score 14.
	30	2.5	Behavior score 19; and evaluation score 12.
	4	2.6	Behavior score 19; and evaluation score 10.
	24	2.7	Behavior score 17; and evaluation score 16 or more.
	7	2.8	Behavior score 17; and evaluation score 14.
	15	2.9	Behavior score 17; and evaluation score 12.
	3	2.10	Behavior score 17; and evaluation score 10.
	42	2.11	Behavior score 15 or less; and evaluation score 14 or more.
58		3.	**Uncertain Need Met, Potential Problem**
	30	3.1	Behavior score 21 or more; and evaluation score 8 or less.
	28	3.2	Behavior score 15 or less; and evaluation score 12 or 10.
30		4.	**Need Unmet, Current Problem**
	15	4.1	Behavior score 19 or less; and evaluation score 8.
	15	4.2	Behavior score 19 or less; and evaluation score 6 or less.
3			**Omitted Due to Missing Data**

Combined Emergency Assistance: Operational Definitions for Need Assessment.

The following definitions are based on the response patterns reported within items 1, 85–90, 93 and 153. The number of cases that is included in each category is presented on the left.

Q1: In general, how is your health now—would you say excellent, good, fair or poor?

Q85: If you were sick, is there someone you could call on to help out around the house, or to help take care of you?

Q86: Who is that? (Get Relationship)

Q87: How available (is/are **Person(s) in Preceding Item**) to help at any particular time if you were sick—always available, often available, sometimes available, available on an emergency basis only.

Q88: In an emergency, is there someone you could call on to get help for you right away?

Q89: Do you have a telephone or not?

Q90: Is there a telephone you can use without going outside?

Q93: Do you generally spend most of the day with someone, or not?

Q153: Are you able to hear on the telephone (when you use the hearing aid)?

Long Term Emergency Assistance

Number		Categories
1044		**1. Need Met, No Apparent Problem**
	634	1.1 Has someone to call if sick (Q85); person is household member (Q86); and person is always available (Q87).
	32	1.2 Has someone to call if sick (Q85); person is household member (Q86); and person is often available (Q87).
	361	1.3 Has someone to call if sick (Q85); person is non-paid and outside of household (Q86); and person is always available (Q87).
	17	1.4 Has someone to call if sick (Q85); person is paid source (Q86); and person is always available (Q87).
115		**2. Need Met, Potential Problem**
	68	2.1 Has someone to call if sick (Q85); person is non-paid and outside household (Q86); and person is often available (Q87).
	9	2.2 Has someone to call if sick (Q85); person is household member (Q86); and person is sometimes available (Q87).
	35	2.3 Has someone to call if sick (Q85); person is non-paid and outside of household (Q86); and person is sometimes available (Q87).
	3	2.4 Has someone to call if sick (Q85); person is a paid source (Q86); and person is often available (87).
143		**3. Uncertain Need Met, Potential Problem**
	3	3.1 Has someone to call if sick (Q85); person is household member (Q86); and person is available on an emergency basis only (Q87).
	24	3.2 Has someone to call if sick (Q85); person is non-paid and outside of household (Q86); and person is available on an emergency basis only (Q87).
	2	3.3 Has someone to call if sick (Q85); person is paid source (Q86); and person is sometimes available (Q87).
	1	3.4 Has someone to call if sick (Q85); person is paid source (Q86); and person is available on emergency basis only (Q87).
	50	3.5 Does not have someone to call if sick (Q85); and lives with others (demographic information from initial screening).
	33	3.6 Does not have someone to call if sick (Q85); lives alone (demographic information from initial screening); and excellent or good perceived health (Q1).
	30	3.7 Does not have someone to call if sick (Q85); lives alone (demographic information from initial screening); and fair perceived health (Q1).
6		**4. Need Unmet, Current Problem**
	6	4.1 Does not have someone to call if sick (Q85); lives alone (demographic information from initial screening); and poor perceived health (Q1).
9		**Omitted Due to Missing Data**

Short Term Emergency Assistance

Number		Categories	
1266		**1. Need Met, No Apparent Problem**	
	865	1.1	Generally spends the day with someone (Q93).
	155	1.2	Does not generally spend the day with someone (Q93); has someone to call in an emergency (Q88); lives with other people (demographic information from initial screening); and has own telephone (Q89).
	245	1.3	Does not generally spend the day with someone (Q93); has someone to call in an emergency (Q88); lives alone (demographic information from initial screening); has own telephone (Q89); and able to hear on telephone (Q153).
	1	1.4	Does not generally spend the day with someone (Q93); has someone to call in an emergency (Q88); lives with others (demographic information from initial screening); does not have own phone (Q89); and has phone to use in building (Q90).
7		**2. Need Met, Potential Problem**	
	6	2.1	Does not generally spend the day with someone (Q93); has someone to call in an emergency (Q88); lives alone (demographic information from initial screening); does not have own phone (Q89); has phone to use in building (Q90); and able to hear on telephone (Q153).
	1	2.2	Does not generally spend the day with someone (Q93); has someone to call in an emergency (Q88); lives with others (demographic information from initial screening); does not have own phone (Q89); and does not have phone to use in building (Q90).
10		**3. Uncertain Need Met, Potential Problem**	
	1	3.1	Does not generally spend the day with someone (Q93); has someone to call in an emergency (Q88); lives alone (demographic information from initial screening); does not have own phone (Q89); does not have phone to use in building (Q90); and able to hear on telephone (Q153).
	4	3.2	Does not generally spend the day with someone (Q93); has someone to call in an emergency (Q88); lives alone (demographic information from initial screening); has own telephone (Q89); and not able to hear on telephone (Q153).
	5	3.3	Does not generally spend the day with someone (Q93); does not have someone to call in an emergency (Q88); and lives with others (demographic information from initial screening).
16		**4. Need Unmet, Current Problem**	
	1	4.1	Does not generally spend the day with someone (Q93); has someone to call in an emergency (Q88);

			lives alone (demographic information from initial screening); does not have own phone (Q89); has phone to use in building (Q90); and not able to hear on telephone (Q153).
	2	4.2	Does not generally spend the day with someone (Q93); has someone to call in an emergency (Q88); lives alone (demographic information from initial screening); does not have own phone (Q89); and does not have phone to use in building (Q90).
	13	4.3	Does not generally spend the day with someone (Q93); does not have someone to call in an emergency (Q88); and lives alone (demographic information from initial screening).
18			**Omitted Due to Missing Data**

Combined Emergency Assistance

Number	Categories
1022	1. **Need Met, No Apparent Problem** Need met, no apparent problem on *both* long term and short term emergency assistance.
116	2. **Need Met, Potential Problem** Need met, potential problem on either long term or short term emergency assistance or on both.
141	3. **Uncertain Need Met, Potential Problem** Uncertain need met, potential problem on either long term or short term emergency assistance or on both.
20	4. **Need Unmet, Current Problem** Need unmet, current problem on either long term or short term emergency assistance or on both.
18	**Omitted Due to Missing Data**

Food Shopping: Operational Definitions for Need Assessment.

The following definitions are based on the response patterns reported within items 133, 135, and 136. The number of cases that is included in each category is presented on the left.

Q133: How much of a problem is shopping for food and other things you need around the house—is it a big problem, some problem or no problem at all?
Q135: Who usually does the grocery shopping? (Get Relationship)
Q136: How often is the food shopping done—would you say it's as often as you'd like, not quite as often as you'd like, or not nearly as often as you'd like?

Number			Categories
1121			1. **Need Met, No Apparent Problem**
	1051	1.1	Food shopping done as often as would like (Q136); and no problem with food shopping (Q133).
	70	1.2	Food shopping done as often as would like (Q136); some problem with food shopping (Q133); and food shopping done by self (Q135).
121			2. **Need Met, Potential Problem**
	24	2.1	Food shopping done as often as would like (Q136); some problem with food shopping (Q133); and food shopping done by household member (Q135).
	15	2.2	Food shopping done as often as would like (Q136); some problem with food shopping (Q133); and food

shopping done by a person outside of household (Q135).

24 2.3 Food shopping done not quite as often as would like (Q136); no problem or some problem with food shopping (Q133); and food shopping done by self (Q135).

5 2.4 Food shopping done not quite as often as would like (Q136); no problem or some problem with food shopping (Q133); and food shopping done by household member (Q135).

7 2.5 Food shopping done not quite as often as would like (Q136); no problem or some problem with food shopping (Q133); and food shopping done by a person outside of household (Q135).

46 2.6 Missing data on whether food shopping done as often as would like (Q136); and no problem or some problem with food shopping (Q133).

53 **3. Uncertain Need Met, Potential Problem**

11 3.1 Food shopping done not quite as often as would like (Q136); and big problem with food shopping (Q133).

10 3.2 Food shopping done not nearly as often as would like (Q136); and no problem or some problem with food shopping (Q133).

31 3.3 Food shopping done as often as would like (Q136); and big problem with food shopping (Q133).

1 3.4 Missing data on whether food shopping done as often as would like (Q136); and a big problem with food shopping (Q133).

8 **4. Need Unmet, Current Problem**

8 4.1 Food shopping done not nearly as often as would like (Q136); and a big problem with food shopping (Q133).

14 **Omitted Due to Missing Data**

Food Preparation: Operational Definitions for Need Assessment.

The following definitions are based on the response patterns reported within items 115–117, 126, 127 and 129. The number of cases that is included in each category is presented on the left.

Q115: Who usually prepares your food? (Get Relationship)

Q116: Is there any reason why you could not prepare your own food? (If Yes, Specify)

Q117: At this present time, is getting the food prepared usually a big problem, a little problem, or no problem at all?

Q126: For a lot of different reasons, people sometimes don't eat the right kinds of food or don't get enough of the foods they should have.

Do you think there are times when you do not eat enough of the right kinds of foods?

Q127: About how often do you not eat enough of the right kinds of foods—would you say you often don't eat the right kinds of foods, or sometimes don't eat the right foods, or just once in a while don't eat the right foods?

Q129: Which of these statements best describes your usual eating pattern:

1. I usually eat regular meals and only rarely snack during the day. (How many regular meals usually—1, 2, or 3?)

2. I usually eat one regular meal a day and snack quite a bit.
3. I usually skip regular meals and just snack all day long.

Number		**Categories**
1110		1. **Need Met, No Apparent Problem**
	818	1.1 Usual eating pattern is 3 regular meals (Q129); eats right kinds of food (Q126); and no problem with food preparation (Q117).
	169	1.2 Usual eating pattern is 2 regular meals (Q129); eats right kinds of food (Q126); and no problem with food preparation (Q117).
	43	1.3 Usual eating pattern is either 2 or 3 meals (Q129); once in a while does not eat right kinds of food (Q127); no problem with food preparation (Q117); *Either* food preparation done by self (Q115) *Or* could prepare own meals (Q116).
	69	1.4 Usual eating pattern is either 2 or 3 regular meals (Q129); eats right kinds of food (Q126); and little problem with food preparation (Q117).
	11	1.5 Usual eating pattern is either 2 or 3 regular meals (Q129); missing data on whether person eats right kinds of food (Q126); and no problem with food preparation (Q117).
125		2. **Need Met, Potential Problem**
	19	2.1 Usual eating pattern is either 2 or 3 regular meals (Q129); eats right kinds of food (Q126); and big problem with food preparation (Q117).
	4	2.2 Usual eating pattern is either 2 or 3 regular meals (Q129); once in a while does not eat right kinds of food (Q127); no problem with food preparation (Q1 7); and cannot prepare own meals (Q116).
	7	2.3 Usual eating pattern is either 2 or 3 regular meals (Q129); once in a while does not eat right kinds of food (Q127); and little problem or big problem with food preparation (Q117).
	47	2.4 Usual eating pattern is 1 regular meal (Q129); *Either* eats right kinds of food (Q126) *Or* once in a while does not eat right kinds of food (Q127); no problem or little problem with food preparation (Q117); *Either* food preparation done by self (Q115) *Or* could prepare own meals (Q116).
	34	2.5 Usual eating pattern is either 2 or 3 regular meals (Q129); sometimes does not eat right kinds of food (Q127); and no problem with food preparation (Q117).
	11	2.6 Usual eating pattern is either 2 or 3 regular meals (Q129); sometimes does not eat right kinds of food (Q127); and little problem with food preparation (Q117).
	3	2.7 Usual eating pattern is either 2 or 3 regular meals (Q129); missing data on whether person eats right kinds of food (Q126); and little problem with food preparation (Q117).
46		3. **Uncertain Need Met, Potential Problem**
	18	3.1 Usual eating pattern is either 2 or 3 regular meals (Q129); often does not eat right kinds of food (Q127);

			and no problem or little problem with food preparation (Q117).
	5	3.2	Usual eating pattern is either 2 or 3 regular meals (Q129); sometimes or often does not eat right kinds of food (Q127); and big problem with food preparation (Q117).
	3	3.3	Usual eating pattern is either 2 or 3 regular meals (Q129); missing data on whether person eats right kinds of food (Q126); and big problem with food preparation (Q117).
	17	3.4	Usual eating pattern is 1 regular meal (Q129); sometimes or often does not eat right kinds of food (Q127); and no problem or little problem with food preparation (Q117).
	3	3.5	Usual eating pattern is 0 regular meals (Q129); eats right kinds of food (Q126); and no problem with food preparation (Q117).
5		**4. Need Unmet, Current Problem**	
	2	4.1	Usual eating pattern is 1 regular meal (Q129); sometimes or often does not eat right kinds of food (Q127); and big problem with food preparation (Q117).
	3	4.2	Usual eating pattern is 0 regular meals (Q129); often does not eat right kinds of food (Q127); and big problem with food preparation (Q117).
31		**Omitted Due to Missing Data**	

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Discussion

Shanas: Could the group that you described as having all their needs currently met and no problems have included a group of severely impaired who are being taken care of by family members?

Branch: Yes, we might have considered those severely impaired elders who are being taken care of by family members to have their needs currently met with no apparent problems. We did differentiate those categorized as need currently met with no apparent problem into three subgroups: (1) Those who were self-sufficient, (2) those who relied on a within household support system (which includes the group you mention), and (3) those who relied on a support system outside their households.

Shanas: As I understood the data, either the people were a lot healthier in Massachusetts than are those in other national studies that have been reported or you are using a different definition of people in need.

Branch: That observation introduces a point I would like to emphasize. This study in Massachusetts is concerned with the elders who have *unmet needs*. They are about 3 to 7 percent of the population, and these people definitely need intensive health care and social support services. We should not neglect these people. The other studies you allude to have focused on a more inclusive concept, namely all the people with needs, regardless of whether the needs are being met or not. I would emphasize that we need to concentrate on the more restricted group, those 3 to 7 percent of the elders with unmet needs who require intensive services, those elders who represent a manageable problem which can be solved by a correct application of existing resources. Those other elders with *met needs*, such as those who have needs and have found someone to help them dress or those who have needs and have a leg brace to help them walk following a stroke, those elders quite likely do not want to be considered as part of the elderly who need help from the social service system. Combining those elders with met needs with those elders with unmet needs to yield one group in need frequently serves to transform a manageable problem into an unmanageable one for the program planners.

In general, the suggestion that the elders in Massachusetts are perhaps demographically different from the national group is not supported by the data, with the exception that the Massachusetts sample underrepresented non-Caucasian elders.

Brehm: A point of clarification in terms of this discussion. Research on disability within the working age population may show that there is a distinction between impairment and working disability where impairment is the physical incapacity to perform and disability is the social incapacity to perform certain required functions.

Part of it relates, then—given a medically definable impairment—to what the expectations of the individual are and what the expectations of the situation are. Someone who is incapable physically of doing much may not define himself as disabled because he had no expectations of being able to do that much. This becomes crystal clear when you are dealing with the working age population where there are many, many more people who are physically impaired than defining themselves as being disabled.

Kovar: What happened to the people who were interviewed the first time and not the second time? Apparently you lost one in eight?

Branch: That is nearly correct. One in eight would be 12.5 percent not reinterviewed, whereas we actually had an 89 percent response rate for the eligible time 2 respondents. Here are the numbers: All 1,625 elderly respondents from time 1 were assumed eligible as time 2 respondents. A total of 146 were subsequently found ineligible as noninstitutionalized elderly in Massachusetts at time 2 (103 had died during the interim, 26 had entered the long-term care institutional system, and 17 had moved out of State). In total, 1,317 personal interviews were conducted with the 1,479 eligible respondents, which is an 89 percent response rate.

Kovar: In your statements on the functional disability and what has happened in the second point in time, it will matter considerably if those who are lost to followup are different from those reinterviewed. That is the only reason I was raising the point. Obviously those who have gone into long-term care facilities may not have an unmet need, but they have a need.

Branch: Yes, but again, the number of people who went into long-term care facilities was too small for us to do anything with statistically at this time. We did collect information on them and they will not be lost.

Kovar: Are you going to reinterview again?

Branch: I hope so.

Siegel: In your calculations of the percentages indicating the probability of dying in 15 months—that is, before the next interview—was age taken into account or controlled for in some way or just ignored?

Branch: Age was a statistically significant discriminator at that first level of analysis, but not at the second level. That is, age categorized into 5-year intervals did not account for 1 percent of R-squared in the regression analysis. Recall that we only had 103 cases distributed over five categories. The fact that age did not reduce R-squared by 1 percent can be an artifact of an insufficient sample size.

Self-Assessment of Physical Function: White and Black Elderly in the United States*

Ethel Shanas, Ph. D.

Department of Sociology
University of Illinois
Chicago Circle Campus
Chicago, IL 60680

In 1972 the Adult Development and Aging Branch of the National Institute of Child Health and Human Development held a conference on the epidemiology of aging. The aim of that conference was to stimulate the use of epidemiologic methods in the study of aging itself. As Adrian Ostfeld (*1*) pointed out in his introduction to the conference papers, aging was not a disease. Like disease, however, aging could be operationally defined, and like disease, aging follows different natural histories in different populations and in different settings. Ostfeld noted that in studying aging using epidemiologic methods one could concentrate on either the correlates or the determinants of the aging process.

The present paper addresses itself to certain of the correlates of aging in two populations: white and black Americans, aged 65 and over, living outside of institutions. As is well known, the life expectancy of white women, the longest-lived group, was 76.6 years compared to a life expectancy of 71.2 years for women of black and other races. For men, the comparable figures were 68.9 years for whites and 62.9 years for blacks and other races. For survivors who reach the age of 65, however, these differences in life expectancy between the races have almost vanished. Life expectancy at age 65 was 13.4 years for white men and 13.4 years for men of black and other races; 17.6 years for white women and 16.7 years for women of black and other races. By age 75, black survivors, on the average, could expect to outlive their white contemporaries (*2*, *3*).

Although life expectancy of persons of black and other races and of whites at ages over 65 is roughly comparable, it cannot be assumed that the physical capacity of these two groups is also comparable. Are the two groups alike in their physical mobility, in their capacity for self-care, and in their reports of illness? Some answers to these questions are given in this paper. Findings are reported in four general areas. These are:

1. The differences in physical mobility between white and black Americans.

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2. The capacity for self-care of these two populations as measured by their self-reports of activities.

3. The relationship between the capacity for self-care and reports of illness requiring as much as a day in bed, and between capacity for self-care and the use of physicians.

4. The relationship between the capacity for self-care and increasing age.

Methods

The data were drawn from a 1975 nationwide probability sample study of the noninstitutionalized population aged 65 and over. The sample used in this survey is so designed that every eligible person, in this instance, every person aged 65 and over not residing in an institution, has a predetermined chance of being located and interviewed.

To insure that black elderly would be represented in the sample in their proper proportion to the total population, the sampling design called for an over-sample of blacks. In the final analysis each case in the sample was weighted by its location. The age and sex characteristics of the sample population agree almost exactly with the characteristics of both the white and black aged populations as reported in the 1970 Census, although there is some slight underrepresentation of older black men in the sample. Findings from the sample, however, may be generalized with confidence to the total elderly population of both races living outside of institutions.

About 5 percent of the white elderly and 3 percent of the black elderly were reported as residents in institutions in the 1970 Census (*4*). The proportion of the elderly living in institutions seems not to have risen between 1970 and 1975. Using data from the Current Population Report, Siegel says: "Slightly less than 5 percent of the population 65 and over resided in institutions in 1975" (*2*).

Findings

Mobility of the aged.—Table III–28 shows the results of a study of the physical mobility of the American aged in 1975. In the total interviewed sample, 14 percent of the aged were classified as being restricted in physical mobility, i.e. bedfast, housebound and able to go outdoors only with difficulty.

In 1972, the National Center for Health Statistics, using somewhat different questions, reported that about 17 percent of the noninstitutionalized elderly had some limitation of their mobility due to a chronic condition. In the National Center analysis, 3 of every 10 of these persons, about 5 percent of all elderly, were described as having limitations sufficiently severe to keep them confined to the house. The remainder, although able to leave the house, needed the assistance of another person or had to use help such as a wheelchair (*5*). In the present 1975 survey, however, persons confined to a wheelchair, who were not totally bedfast, were classified as housebound. About 7 percent

Table III-28.

Physical mobility of the noninstitutional population aged 65 and over by sex and race: 1975 (percentage distribution)

Degree of mobility	Whites			Blacks			Total [1]		
	Men	Women	All	Men	Women	All	Men	Women	All
Sample located: [2]									
Bedfast	3	3	3	6	3	4	2	3	3
Housebound	6	8	7	5	10	8	6	8	7
Ambulatory	91	89	90	89	87	88	92	89	90
Total	100	100	100	100	100	100	100	100	100
N (weighted)	(2, 458)	(3, 573)	(6, 039)[3]	(218)	(325)	(545)[3]	(3, 070)	(4, 484)	(7, 660)[3]
Sample interviewed:									
Bedfast	2	2	2	2	1	1	2	2	2
Housebound	4	6	5	1	7	5	3	6	5
Ambulatory	95	91	93	97	92	94	95	91	93
Can go outdoors with difficulty	4	8	6	5	17	12	4	8	7
Can go outdoors without difficulty	91	84	86	92	75	81	91	83	86
Total	100	100	100	100	100	100	100	100	100
N (weighted)	(2, 127)	(3, 140)	(5, 267)	(177)	(286)	(463)	(2, 314)	(3, 441)	(5, 755)

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[1] The total column includes persons of races other than white and black (25 weighted cases); it also includes 1,051 weighted cases where race is not known.

[2] The percentage of bedfast respondents is based on all bedfast persons located. Interviews with proxy respondents were taken for all bedfast persons who could not be interviewed. The percentages of housebound and ambulatory respondents in the sample located were estimated from their proportions in the sample interviewed, and from background data on nonrespondents secured by the interviewers.

[3] Includes cases where sex is not known: whites, 8 weighted cases, blacks, 1 weighted case; and total, (whites, blacks and unknown races) 105 weighted cases.

of all persons interviewed in the present survey had mobility limitations so severe that they were classified as either bedfast or housebound.

These reports of limitations of physical mobility among the elderly population of the United States as a whole obscure the restrictions on physical mobility reported by the black aged, particularly black women. Eighteen percent of all blacks compared with 13 percent of all whites were restricted in their physical mobility. Among black women, 25 percent were restricted in their physical mobility; i.e., one of every four aged black women were either bedfast, housebound, or able to go outdoors only with difficulty.

Capacity for self care.—An index score to measure the functional capacity of black and white Americans is used in tables III–29, III–30, and III–31. This index differs from the measure of sociobiological functioning developed by Sidney Katz and his associates, the well-known index of ADL, which is primarily concerned with assessments of deficits in function among sick people (*6*). Instead, the index focuses on what may be called "degree of fitness" to derive some measure of the ability of the aged person to perform those tasks which make him independent of others for personal care (7). The scores are based on the old person's answer to six questions: (1) Can he go out-of-doors? (2) Can he walk up and down stairs? (3) Can he get about the house? (4) Can he wash and bathe himself? (5) Can he dress himself and put on shoes? And finally (6) Can he cut his own toenails? Each respondent is asked whether he can do each of these tasks without difficulty and without assistance, with some difficulty, but still without the help of another person, and finally, with difficulty and only with the help of another person. These questions on the physical functioning of the aged designed to measure capacity for self-care have been used in national studies of the elderly in Belgium, Britain, Denmark, Israel, Poland, and Yugoslavia as well as in the United States and their validity and reliability are well-established (*8*, *9*). Scores for functional capacity may range from zero or no reported limitations, to seven or more. The higher the index the greater the incapacity.

While only 12 percent of all aged Americans have scores of three or more, which indicate some major limitations in their capacity for self-care, when this gross total is analyzed by race, 11 percent of whites have scores of three or more compared to 21 percent of blacks. Women, irrespective of race, are more likely than men to be restricted in their capacity for self-care. Again, black women are the most likely of all persons to report limitations on their capacity for self-care. One in every four of these women have scores as high as three or more (See table III–29).

Table III–30 gives the proportion of the elderly of each race who report that they have difficulty with or are unable to perform the various physical tasks that make up the index of functional capacity. From this table it is obvious that among these tasks, two are especially difficult for the American aged. These are "walking stairs" and "cutting toenails." Again, the differences between white and black elderly

Table III–29.

Capacity for self-care, noninstitutional population aged 65 and over by sex and race: 1975[1] (percentage distribution)

Index Score	Whites			Blacks			Total		
	Men	Women	All	Men	Women	All	Men	Women	All[2]
0	74	65	69	59	34	44	73	62	67
1 to 2	18	21	20	29	38	35	19	23	21
3 to 4	5	6	6	7	10	9	5	7	6
5 to 6	2	5	4	5	11	8	2	6	4
7 or more	([3])	2	1	([3])	7	4	([3])	3	2
Total	100	100	100	100	100	100	100	100	100
N (weighted)	(2, 089)	(3, 071)	(5, 160)	(175)	(283)	(458)	(2, 274)	(3, 366)	(5, 640)

[1] Excludes bedfast persons.

[2] The total column includes persons of races other than white and black (11 weighted cases); and 14 weighted cases where race is not known.

[3] Less than 1 percent after rounding.

are apparent. Twice as many blacks as whites have problems in walking stairs, and almost twice as many blacks as whites have problems in cutting their toenails. Almost one in every four whites and two in every four blacks find stairs a problem; one in every five whites and one in every three blacks has problems with his toenails. The other items in this index, getting about the house, washing and bathing, dressing and putting on shoes, are less difficult for older Americans, but in every instance twice as many blacks as whites report that they have difficulty in performing these common tasks. It should perhaps be noted that, as indicated by the reported problem with toenail cutting, problems of foot care are mentioned so often by old people that it is difficult to understand how broad-spectrum podiatry services continue to be excluded from coverage under health insurance for the aged.

There is a clear and expected relationship between index scores of functional capacity and old peoples' reports that they have spent at least one day in bed because of illness during the past year, and that they have recently seen a doctor. Table III–31 shows these relationships for all elderly and for whites and blacks separately. One of every four old people in the United States reported that they had spent at least a day in bed because of illness during the year before they were interviewed. Those persons whose incapacity scores were three or more, however, were twice as likely to have spent time in bed as persons with lower index scores. Again, the difference between the two racial groups is apparent. Blacks were almost twice as likely as whites to report some time in bed because of illness. For all older people, higher index scores were associated with greater recent use of physicians. Blacks were more likely than whites to report a recent doctor visit. About 4 of every 10 blacks compared to 3 of every 10 whites reported such a visit.

Increasing age and capacity for self-care.—As shown in table III–32, capacity for self-care was found to decline with the age of the sample cohort. At ages 65 to 69, only one person in 14 had an index score of three or more. Among those age 80 and over, however, one person in four had an index score of three or more on the capacity scale. Blacks start out at ages 65 to 69 with higher index scores than whites and these higher scores for blacks continue for each age group. However, the older the cohorts of blacks and whites, the less the differences in capacity scores between them. For ages 65 to 69, blacks are three times as likely as whites to have high scores; for ages 80 and over, only 1½ times as likely. The decline in the differences in high scores between blacks and whites for advanced age cohorts can be correlated with the decline in the differences in life expectancy between the two races as survivors age. One may speculate that, were valid reports available for persons aged 90 and over, blacks would have the same or better reports of physical function than their white contemporaries.

Table III–33 shows the proportion of persons of different ages reporting difficulty with each of the items that make up the index of capacity. The proportion of persons who report they have difficulty with a specific task increases with age for both whites and blacks. At

Table III–30.

Percentage of persons aged 65 and over reporting difficulty with common physical tasks by sex and race: 1975 [1]

Task	Percent with difficulty								
	Whites			Blacks			Total [2]		
	Men	Women	All	Men	Women	All	Men	Women	All
Walking stairs	16	26	22	32	51	44	18	28	24
Getting about the house	3	9	6	5	18	13	3	9	7
Washing, bathing	4	10	7	7	18	14	4	10	8
Dressing, putting on shoes	6	8	7	6	20	15	6	9	8
Cutting toenails	17	22	20	21	44	36	17	24	21
N (weighted)	(2, 089)	(3, 071)	(5, 160)	(174)	(284)	(458)	(2, 273)	(3, 367)	(5, 640)

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[1] Excludes bedfast persons; includes housebound persons who cannot do physical tasks at all.

[2] The total column includes persons of race other than white and black (11 weighted cases) and 14 weighted cases where race is not known.

Table III–31.

Illness and use of doctors, persons aged 65 and over by capacity scores and race: 1975 [1]

Illness, use of doctors	Whites			Blacks			All [2]		
	0 to 2	3+	All	0 to 2	3+	All	0 to 2	3+	All
Percent ill in bed in past year	22	47	24	33	64	40	22	50	26
N (weighted)	(4, 576)	(585)	(5, 160)	(358)	(99)	(458)	(4, 956)	(683)	(5, 640)
Percent who saw doctor within past month	30	47	32	38	60	43	30	49	33
N (weighted)	(4, 568)	(582)	(5, 150)	(356)	(99)	(456)	(4, 946)	(680)	(5, 627)

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[1] Excludes bedfast persons.

[2] The totals in these columns includes races other than white and black (11 weighted cases); and 14 weighted cases where race is not known.

Table III–32.

Capacity for self-care by age, sex, and race, noninstitutional population aged 65 and over: 1975[1] (Percentage distribution)

Degree of mobility	Whites			Blacks			All[2]		
	Men	Women	All	Men	Women	All	Men	Women	All
65 to 69:									
0 to 2	94	94	94	92	77	83	93	92	93
3 plus	6	6	6	10	23	18	6	8	7
Total	100	100	100	100	100	100	100	100	100
N (weighted)	(804)	(1, 107)	(1, 911)	(73)	(113)	(186)	(887)	(1, 220)	(2, 107)
70 to 74:									
0 to 2	94	88	90	(3)	78	86	94	87	90
3 plus	6	12	10	(3)	22	14	6	13	10
Total	100	100	100	(3)	100	100	100	100	100
N (weighted)	(615)	(848)	(1, 463)	(47)	(68)	(115)	(662)	(920)	(1, 582)
75 to 79:									
0 to 2	93	82	86	(3)	59	70	92	80	85
3 plus	7	18	14	(3)	39	29	8	20	15
Total	100	100	100	(3)	100	100	100	100	100
N (weighted)	(379)	(533)	(912)	(28)	(51)	(79)	(407)	(591)	(998)
80 and over:									
0 to 2	87	74	77	(3)	67	65	83	73	77
3 plus	15	26	22	(3)	33	35	17	27	23
Total	100	100	100	(3)	100	100	100	100	100
N (weighted)	(291)	(583)	(874)	(27)	(51)	(78)	(318)	(634)	(952)

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[1] Excludes bedfast persons.

[2] The totals in these columns include persons of races other than white and black (11 weighted cases); and 14 weighted cases where race is not known.

[3] Percentages not computed where base is less than 50.

Table III–33.

Percentage of persons aged 65 and over reporting difficulty with common physical tasks by age, sex, and race: 1975 [1]

Age and task	Percent reporting difficulty [2]					
	Whites			Blacks		
	Men	Women	All	Men	Women	All
65 to 69:						
Walking stairs	15	15	15	27	44	37
Getting about the house	4	4	4	3	13	9
Washing and bathing	4	6	5	5	14	10
Dressing, putting on shoes	4	5	5	6	21	15
Cutting toenails	11	10	10	15	40	30
N (weighted)	(804)	(1, 107)	(1, 911)	(73)	(113)	(186)
70 to 74:						
Walking stairs	15	23	19	(3)	52	42
Getting about the house	1	7	4	(3)	15	10
Washing and bathing	3	7	5	(3)	12	8
Dressing, putting on shoes	3	6	5	(3)	10	8
Cutting toenails	19	20	19	(3)	37	28
N (weighted)	(615)	(848)	(1, 463)	(47)	(68)	(115)
75 to 79:						
Walking stairs	14	34	25	(3)	58	50
Getting about the house	2	10	6	(3)	26	19
Washing and bathing	4	12	8	(3)	24	19
Dressing, putting on shoes	6	10	8	(3)	22	17
Cutting toenails	20	27	24	(3)	48	41
N (weighted)	(379)	(533)	(912)	(28)	(51)	(79)
80 plus:						
Walking stairs	26	46	39	(3)	60	58
Getting about the house	5	17	13	(3)	25	19
Washing and bathing	7	18	14	(3)	29	26
Dressing, putting on shoes	14	16	16	(3)	28	23
Cutting toenails	25	42	37	(3)	58	54
N (weighted)	(291)	(583)	(874)	(27)	(51)	(78)

---

[1] Excludes bedfast persons; excludes races other than white and black (11 weighted cases); and 14 weighted cases where race is not known.

[2] Rates computed on the basis of the number of eligible respondents who answered each of these questions. The base for each rate therefore varies slightly. The number of "no answers" ranged from 0 to 3; in no instance did less than 99 percent of the respondents answer a question.

[3] Percentages not computed where base is less than 50.

least 1 of every 10 whites and 2 of every 10 blacks aged 80 and over has problems getting about the house, washing and bathing, and dressing and putting on shoes. The more common complaints—problems with stairs, problems with cutting toenails—are reported by 4 of every 10 whites and 5 to 6 of every 10 blacks in this age group.

Table III–34 gives the relationship between the age of older people and their reports that they have spent at least 1 day in bed because of illness during the past year, and their reports of their visits to doctors. In interpreting this table, it should be recalled that the most physically deteriorated and frail old people, usually aged 80 plus, are likely to be institutionalized and thus excluded from this study. It is perhaps for this reason that there is no marked increase with age in the proportion of persons who said that they had spent at least a day in bed because of illness during the previous year. Similarly, among whites there is no marked increase with age of those who said they had seen a doctor during the month before their interview. Blacks are less likely to be institutionalized than whites. The reports for blacks show the expected increase with age in the proportion who have seen a doctor during the past month. Women, whether white or black, are more likely than men to report that they have seen a doctor. Once again, black women are more likely than white women to be ill and more likely than whites to report that they have seen a physician.

Summary and Future Research

The findings from a national survey of older Americans show that black Americans are more restricted than whites in physical mobility; that black Americans are less able than whites to care for their physical needs; that blacks are more likely than whites to have illnesses which keep them in bed; and that blacks are more likely than whites to report recent contacts with a doctor. Black women appear to be the most incapacitated group in the noninstitutionalized aged population, those most likely to be restricted in mobility and those most in need of help in the activities related to self-care.

With advances in age, the physical differences between black and white cohorts appear to become less, but at least 1 of every 10 whites and 2 of every 10 blacks in the group over 80 has problems in getting about the house, washing and bathing, and dressing and putting on shoes.

The data in this report point to at least two important areas for both research and policy consideration. The first is: What can be done to improve the physical functioning of older Americans? About one-fifth of the population over 65 is now over 80 (4.5 million persons). By the year 2000, it is expected that those over 80 will total 6 million persons. If there is no major change in the capacity for self-care among this segment of the aged during the next two decades, the demands for home helpers and home health aids to assist the over-eighties in the activities of daily living will be enormous.

These demands for help will be further increased by changes in family structure and size, and by the increased labor force participation

Table III-34.

Illness and use of doctors, persons aged 65 and over, by age, sex, and race: 1975 [1]

Illness, use of doctors	Men					Women					All				
	65-69	70-74	75-79	80+	All	65-69	70-74	75-79	80+	All	65-69	70-74	75-79	80+	All
Percent ill in bed in past year:															
Whites	22	22	19	22	21	30	23	25	26	27	27	23	22	25	24
N (weighted)	(804)	(615)	(379)	(291)	(2,039)	(1,107)	(848)	(533)	(583)	(3,071)	(1,911)	(1,463)	(912)	(874)	(5,160)
Blacks	34	(2)	(2)	(2)	33	49	41	38	44	44	43	37	37	40	40
N (weighted)	(73)	(47)	(28)	(27)	(175)	(113)	(68)	(51)	(52)	(284)	(186)	(115)	(79)	(79)	(459)
Percent who saw doctor within past month:															
Whites	29	31	31	33	30	31	32	39	32	33	30	32	36	32	32
N (weighted)	(804)	(609)	(379)	(291)	(2,083)	(1,107)	(848)	(532)	(580)	(3,067)	(1,911)	(1,457)	(911)	(871)	(5,150)
Blacks	19	(2)	(2)	(2)	35	47	44	64	30	48	36	47	55	42	43
N (weighted)	(73)	(47)	(28)	(27)	(175)	(112)	(68)	(50)	(52)	(282)	(185)	(114)	(78)	(79)	(457)

---

[1] Excludes bedfast persons; excludes races other than white and black (11 weighted cases); and 14 weighted cases where race is not known.

[2] Percentages not computed where base is less than 50.

of women who to this stage have acted as unpaid family helpers in the care of the elderly (*10*). There is an obvious need to decrease the incidence and severity of disabling conditions among the aged. All possible means of education, prevention and treatment should be explored in an effort to achieve these goals (*11*).

Another finding of this research has more immediate applications. It is apparent that reports of the health status of the elderly which group all Americans together obscure the special health problems of minority groups, blacks and probably others. It is obvious that the health problems of blacks and whites differ, but what then of aged Americans of other ethnic groups within the American scene? We need to disaggregate summary data in order to know more about the health situation of special minority groups among the aged, and more about the health status of older Americans of different ethnic backgrounds and cultures. By comparing these groups, we can learn which differences in physical functioning in old age appear to be the result of different nationality and cultural backgrounds and which appear to be primarily age-related.

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Discussion

Adelman: With regard to your functional index, Dr. Shanas, how can you make distinctions between actual functional loss and other parameters such as lack of desire for self-care, desire for interaction with other people, or practices of the physicians that they choose?

Shanas: There are a series of questions in the index which serve to define the categories. In the case of the bedfast, they absolutely do not get out of bed. Whether this is because they do not want to get out of bed or it is too cold or for some other reason, I cannot say. In the case of the housebound, the respondent has to have been in the house for at least 6 weeks. There are a series of questions about that. These questions have been used in national studies throughout the world, and apparently, they do discriminate for gross categories. People behave quite differently if they are bedfast or housebound. If anyone does not want to do something and says he cannot do it, then effectively he cannot do it.

Gruenberg: I think there are three different questions involved in that problem. One is, does the person do it; the second is, can he; the third is, what does he say? I do not think you can actually separate the results of your questions in describing who can get out of bed and who cannot. Our center interviewed some elderly and they say, "I am bedbound or housebound," and when asked why, they say "the doctor told me." Actually, in a questionnaire, I do not think there is any way of distinguishing the sensation of being housebound from being really housebound. Also, you do not know what they did last week, last month, or last year.

Shanas: I first started to use some of these interviews as far back as 1956 when we interviewed a national sample of older people and a national sample of persons other than spouses who were taking care of them. This was a validation study. When people say they absolutely cannot do something, a number of them say that no one helps and that they do it themselves.

I would not say that, for any one person, this index is predictive, but for gross categories, it works very well. This group might be interested in something that Sidney Katz and I are considering jointly. Dr. Katz, together with Austin Chinn and others, did the original work on the development of an index of the activities of daily living. He has had some demonstration studies in Michigan where he is trying to figure out different packages for use among people who need home care or are getting home care.

We are trying to work out a mini-index of activities of daily living that will compare with my data and use his findings to make national projections on our data. His research is interesting and informative. We hope to use this national study to make some national cost predictions for various service packages.

Branch: What were your sample sizes for each category?

Shanas: We used a complicated weighting scheme in which we had different sampling ratios for census tracts, depending on their proportion of blacks. We also used a weighting scheme for individual households. In the end, the unweighted sample included almost 500 blacks and about 1,600 whites, for a total population of 2,100 interviewed. The weighted population was 5,743. This is one of the largest samples of older blacks available for this kind of analysis.

Branch: The screening task must have been tremendous.

Shanas: Yes, the screening task was. Although I do not have the material here, I believe we screened about 12,000 households.

Branch: Looking at table III–29, I do not see how your index score is indicated. You said that the 12 percent of the elderly with index scores of 3 or more were a target group. Have many of these people perhaps solved their problem? As long as they have solved it, they don't have an unmet need. There are times when it appears that the elderly have more problems than they actually do. I think this is part of the point I was trying to make with some of my data.

Shanas: If you recall, persons who could only perform the tasks with difficulty and only with the help of another person were scored higher. I think some persons have solved their problems. When you ask them who helps with this task that they cannot do at all, it turns out that the individual does it for himself. But many of them have not solved this. What we are trying to do, and I know what you are trying to do in Massachusetts, is to zero in on the individuals in the target group.

Branch: I found one other interesting thing by asking a followup question. If a person was receiving help from another person, we asked if he or she was getting enough help. We found that about half of the people who were receiving help said that they needed even more than they were getting. In most other studies, if a person is receiving help, he or she is automatically categorized in the group of having solved his or her problems.

Gruenberg: I would like to ask whether you would agree that when you start a study like this, you want to find a target population of these services? At other times, you are interpreting your data about the frequency of these phenomena in blacks and whites. If you really want to know about frequency phenomena, you should not exclude the institutionalized population.

You should go about this study somewhat differently, if you want to get your target population. It seems to me that if you focus on unmet needs, you have to focus on the data gathering point at the sampling point. You did not take institutionalized populations, presumably, because you are trying to find unmet needs in the community.

Shanas: Yes.

Gruenberg: Your data showed a much higher prevalence of need among blacks. Perhaps blacks have more problems than whites outside the institution. Maybe the difference was made up by the proportion of blacks and whites in your study.

Shanas: Not really. The differences are not so vast.

Gruenberg: Yes, but the problems are much more severe in nursing homes.

Shanas: No, not necessarily.

Brehm: When you are doing a study with a small sample, you can talk about those who are in an institution and those who are outside the institutions. When you are doing a major nationwide survey, the sampling problems are so enormous in trying to coordinate an institutionalized and a noninstitutionalized population that they are traditionally done in two different studies.

Shanas: We need the National Center for Health Statistics to do the institutional study.

Costa: Dr. Shanas, you talked about the Cornell medical index yielding some information on functional health status. Would it be more accurate to say that your measure and Dr. Branch's measure should be termed self-reported capacity as opposed to functional capacity?

Shanas: I call it functional capacity.

Costa: The distinction I am trying to get at is self-reported capacity rather than actual, measurable function.

Kovar: I want to respond to Dr. Gruenberg's question. He is right that we have to look for comparability in data collection in that we survey and interview the noninstitutionalized population using a different set of procedures and questionnaires than we use for the institutionalized population. There is no argument about that.

However, the problem of lacking comparable data for the entire target population is greater than that. The National Center for Health Statistics does not collect data in any long-term institutions, except in the nursing homes. There are elderly people in other long-term institutions, State and county mental hospitals, VA hospitals, homes for the retarded, and homes for the aged and we have very little information about their needs for care.

One of the problems is the lack of a good sampling frame. Another problem which you may not be aware of is that even though we do surveys in the community population and in the nursing homes, we do not have permission to actually interview the residents of the homes. Therefore, reporting in the community is primarily self-reporting or family reporting. In the nursing homes, it is an administrator, a nurse, or someone else reporting on the resident. So that is another data problem. It makes it extremely difficult even when we want to put the two segments together. We don't know whether we are getting the same kind of information.

Technically, it is possible to collect comparable data. Administratively, it is difficult. But you are right, Dr. Gruenberg. However, I do not think the difference in the proportion of black and white elderly people who are in institutions would change Dr. Shanas' conclusions. Including the institutionalized population might affect the magnitude of the differences; I don't think they would be eliminated.

Closing Remarks by Chairperson Judith Cohen, Ph. D.

I think that it is only in research like this, which combines levels of analysis, that we can begin to understand how responses to the environmental circumstances described fit together. I was somewhat surprised by some of Dr. Kasl's findings that, in fact, things did not necessarily fit together in the same people or the expected pattern. For example, hospitalization experience was not necessarily associated with reporting of negative psychological outcomes. As long as we look at these outcomes one at a time, we may have no problem statistically, but we may not have much insight either.

Another theme of importance for further research might be illustrated by an alternative interpretation of your findings on the interviewer's assessment of how people were doing. What we are really trying to understand is a process of adaptation. We are much better at identifying impingements that we think require adaptation, than we are measuring or understanding the process of coping with them. I just wonder if the interviewer was seeing things about coping that the subject could not necessarily express accurately. I do not know; it is a guess, no more than that. But I think that side of the process remains to be developed and the descriptive measures worked on much more than we have up to now.

Session IV

Demographic Trends and Health Care Implications

Opening Remarks by Chairperson Don C. Gibson, D.V.M., M.P.H.

In this session, discussion will center around an information base for the application of epidemiologic methods. This base includes the expected demographic changes of the aging population, their need for and utilization of health care and the financing of this health care.

Recent and Prospective Demographic Trends for the Elderly Population and Some Implications for Health Care

Jacob S. Siegel*

Population Division
U.S. Bureau of the Census
Washington, D.C. 20233

Although the aging process goes on steadily throughout life, the term is commonly employed to refer to the changes in later life, following the reproductive age period. Aging proceeds at different rates for different individuals if we define it in physiological or functional terms rather than merely chronological terms. For some, the signs of physiological deterioration or the ability not to function independently come earlier than for others, but they inevitably appear for all as time passes. Demographically, however, aging is defined essentially in terms of chronological age, on the assumption that for large populations the aging process, functional age, and physiological age follow chronological age closely.

For convenience and simplicity, in the present discussion of the demography of aging, the age range 65 and over as a whole is employed as the principal basis for identifying and describing the elderly population (*1*). It is important to note, however, that the older population is not a demographically homogeneous group. Although there is the same chronological "distance" between ages 65 and 85 as there is between 45 and 65, the demographic "distance" is quite different. If we compare the population 80 and over with the population 60 to 69, for example, we find many sharp differences with respect to such characteristics as health, living arrangements, marital status, work status, income, education, kinship support, and use of leisure time.

Number and Proportion of Elderly Persons

The age group 65 and over is expected approximately to double in size between 1976 and 2020, moving from 23 million in 1976 to 32 million in 2000 to 45 million in 2020 (table IV–1) (*2*). In this period, all age segments of the elderly population are expected to grow rapidly, particularly the extreme aged. We should have about 17 million persons 75 and over and about 5 million 85 and over in the year 2020. The accuracy of projections of population *numbers* for the older age groups is dependent largely on the accuracy of the projections of mortality, particularly at the older ages; the projections of population numbers do not depend upon future projections of births. This suggests that we can have a fair degree of confidence in the general level of the population and the growth rates shown.

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*The views expressed in this paper do not necessarily reflect the position of the U.S. Bureau of the Census.

Table IV-1.

Total population in the older ages and decennial increases: 1950 to 2040

(Numbers in thousands. Estimates and projections as of July 1. Figures refer to the total population of the 50 States and District of Columbia. A minus sign (—) denotes a decrease. See text for explanation of series I, II, and III.)

Year	60 years and over			65 years and over			70 years and over			75 years and over			85 years and over		
		Increase in preceding decade			Increase in preceding decade			Increase in preceding decade			Increase in preceding decade			Increase in preceding decade	
	Number	Amount	Percent	Number	Amount	Percent	Number	Amount	Percent	Number	Amount	Percent	Number	Amount	Percent
ESTIMATES															
1950	18,500	(1)	(1)	12,397	(1)	(1)	7,348	(1)	(1)	3,904	(1)	(1)	590	(1)	(1)
1960	23,828	5,328	28.8	16,675	4,278	34.5	10,394	3,046	41.5	5,621	1,717	44.0	940	350	59.3
1970	28,753	4,925	20.7	20,087	3,412	20.5	13,065	2,671	25.7	7,600	1,979	35.2	1,432	492	52.3
1976	32,244	(1)	(1)	22,934	(1)	(1)	14,654	(1)	(1)	8,741	(1)	(1)	1,966	(1)	(1)
PROJECTIONS [2]															
1980	34,724	5,971	20.8	24,927	4,840	24.1	16,227	3,162	24.2	9,434	1,834	24.1	2,294	862	60.2
1990	40,184	5,460	15.7	29,824	4,897	19.6	19,803	3,576	22.0	12,021	2,587	27.4	2,881	587	25.6
2000	41,973	1,789	4.5	31,822	1,998	6.7	22,630	2,827	14.3	14,386	2,365	19.7	3,756	875	30.4
2010	49,850	7,877	18.8	34,837	3,015	9.5	23,211	581	2.6	15,060	674	4.7	4,575	819	21.8
2020	63,265	13,415	26.9	45,102	10,265	29.5	29,126	5,915	25.5	16,975	1,915	12.7	4,776	201	4.4
2030	70,737	7,472	11.8	55,024	9,922	22.0	37,936	8,810	30.2	23,170	6,195	36.5	5,681	905	18.9
2040 (II)	69,806	−931	−1.3	54,925	−99	−0.2	40,774	2,838	7.5	27,907	4,737	20.4	7,980	2,299	40.5
Range (III)	68,566	−2,171	−3.1												
Range (I)	71,445	708	1.0												

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[1] Not applicable.

[2] Base date of projections is July 1, 1976.

Source: U.S. Bureau of the Census, "Current Population Reports," series P-25, nos. 311, 519, 614, 643, and 704.

When we consider the *proportion* of elderly persons in the total population, however, we can have much less confidence in the figures. They are directly affected by projections of the total population, and hence of fertility, as well as by projections of the elderly population. It seems likely now, more likely than it did a few years ago, that the proportion of the aged in the total population will continue to rise. The rise may not be a steady one because of the probable fluctuations in future fertility, but two of the three series of projections published by the Census Bureau (those having lower fertility) show a fairly steady rise in the proportion and all of the projected range for the proportion is above the level of 1976.

If fertility moves toward replacement level (fertility somewhat above the level of 1976) between 1976 and 1990 (series II projections) (*3*), we can expect 15.5 percent of the population to be aged 65 and over in the year 2020 as compared with 10.7 percent in 1976 (table IV–2 and figure IV–1). The proportions 65 and over corresponding to the population projections based on below-replacement fertility (series III) and above-replacement fertility (series I) are 17.8 and 12.7 percent, respectively. As fertility decreases, the proportion of the aged tends to increase. The series of projected percents 65 and over to 2020, including the projected range, are as follows:

Year:	Percent	
1976	10.7	
1980	11.2	(11.1–11.3)
1990	12.2	(11.7–12.6)
2000	12.2	(11.1–12.9)
2010	12.7	(11.1–13.9)
2020	15.5	(12.7–17.8)

If fertility continues at current low levels and mortality declines moderately, as is assumed, the percentage of elderly persons can be expected to be close to the highest percentage, 18 percent, for the year 2020 shown in the most recent set of population projections. A sharp rise may occur in the 2010–2020 decade because of the entry of the 1945–60 baby-boom cohorts into the aged population. There may be only a modest rise in the proportion after 2020, and particularly after 2030, as a result mainly of the decline in the number of births after 1960.

Fertility is expected to be a more important determinant of shifts in the age structure of the population than mortality. Both recent changes in the birth rate and shifts in the numbers of births 65 years earlier jointly affect the proportion of elderly. Large reductions in mortality occurring mainly at the older ages could contribute to moderate increases in the percentage of the elderly, however. If, on the other hand, fertility rises and mortality declines very little at the older ages in future years, as is possible, the proportion of elderly persons in the population may stop rising and may even decline.

The older population has generally been getting older and is expected to continue doing so for a few decades. For example, the proportion of persons in the age group 65 and over who are 75 and over is now rising and will generally continue to rise until at least the year

Table IV-2.

Percent of the total population in the older ages, by sex: 1950 to 2020

(Estimates and projections as of July 1. Based on totals including Armed Forces overseas. See text for explanation of series I, II, and III)

Age and sex	1950	1960	1970	1976	Projections[1]					
					1980		2000		2020	
					II	Range I–III	II	Range I–III	II	Range I–III
ALL RACES										
Both sexes:										
60 years and over	12.1	13.2	14.1	15.0	15.6	15.5–15.7	16.1	14.8–17.1	21.8	17.9–25.0
65 years and over	8.1	9.3	9.9	10.7	11.2	11.1–11.3	12.2	11.3–12.9	15.5	12.7–17.8
70 years and over	4.8	5.8	6.4	6.8	7.3	7.2– 7.4	8.7	8.0– 9.2	10.0	8.2–11.5
75 years and over	2.6	3.1	3.7	4.1	4.2	4.2– 4.3	5.5	5.1– 5.9	5.9	4.8– 6.7
85 years and over	0.4	0.5	0.7	0.9	1.0	1.0– 1.0	1.4	1.3– 1.5	1.6	1.3– 1.9
Male:										
60 years and over	11.8	12.4	12.6	13.1	13.6	13.5–13.7	13.8	12.7–14.7	19.3	15.7–22.3
65 years and over	7.7	8.5	8.5	9.0	9.3	9.3– 9.4	10.0	9.2–10.7	13.2	10.7–15.2
70 years and over	4.5	5.2	5.3	5.5	5.8	5.7– 5.8	6.8	6.2– 7.2	8.0	6.5– 9.2
75 years and over	2.3	2.7	3.0	3.1	3.1	3.1– 3.2	4.0	3.7– 4.2	4.3	3.4– 4.9
85 years and over	0.3	0.4	0.5	0.6	0.7	0.6– 0.7	0.8	0.8- 0.9	0.9	0.8– 1.1
Female:										
60 years and over	12.5	14.1	15.6	16.8	17.6	17.4–17.7	18.3	16.9–19.3	24.1	20.0–27.5
65 years and over	8.6	10.0	11.2	12.3	13.0	12.9–13.1	14.3	13.2–15.1	17.8	14.7–20.3
70 years and over	5.2	6.3	7.5	8.1	8.8	8.7– 8.8	10.5	9.7–11.1	12.0	9.9–13.6
75 years and over	2.8	3.5	4.4	5.0	5.3	5.3– 5.3	7.0	6.5– 7.4	7.4	6.1– 8.4
85 years and over	0.5	0.6	0.9	1.2	1.4	1.4– 1.4	2.0	1.9– 2.1	2.3	1.9– 2.6

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[1] Base date of projections is July 1, 1976.

Source: U.S. Bureau of the Census, "Current Population Reports," series P-25, nos. 311, 519, 614, 643, and 704.

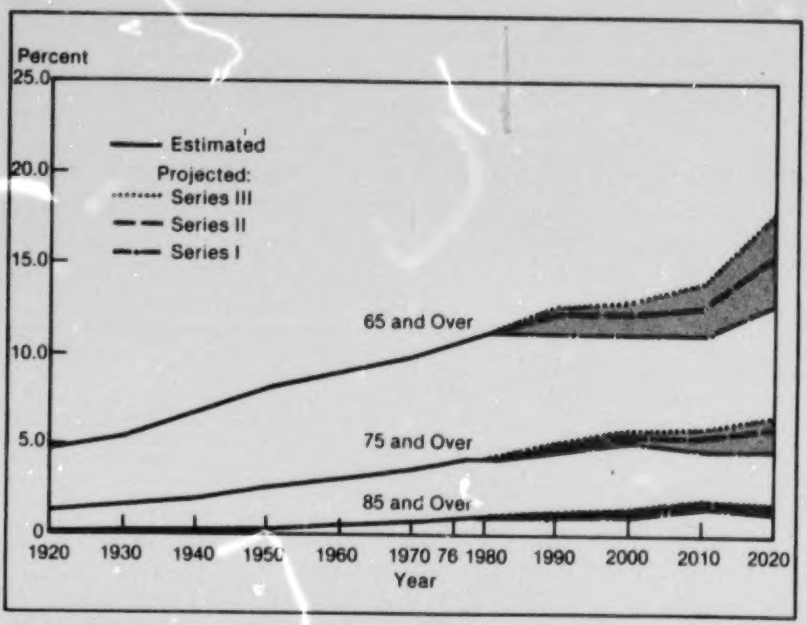

Figure IV–1.

Percent of the total population at the older ages: 1920 to 2020.

Note: Points are plotted for years ending in zero except for 1976.

Source: Table IV-2 and *Current Population Reports*, series P-25, Nos. 311, 519, 614, 643, and 704.

2000. In 1976 the proportion was 38 percent. In the year 2000, we can expect the proportion to be about 45 percent (table IV–3). It is then likely to fall back to about 38 percent again in 2020 as the larger cohorts born in the high fertility period following World War II enter the younger segment of the group, 65 to 74 years.

Aged Dependency Ratios

The ratio of the number of elderly persons (65 and over) to the number of persons of the usual working ages (18 to 64), a rough reflection of the balance of elderly "dependents" to "producers," more than doubled between 1920 and 1960. It has been barely rising since then and is expected to continue increasing slowly in the next several decades (table IV–4). The ratio was 18 per 100 persons 18 to 64 in 1976, may rise slightly to 20 by 1990, where it may remain for a few decades, and then should rise sharply (26 in 2020, series II) as the postwar birth cohorts reach 65 years of age.

At present, about three-quarters of the persons 65 and over have at least one living child, but only a small proportion of elderly persons

Table IV-3.

Percent distribution of the population 65 years and over by age: 1950 to 2020

Age	1950	1960	1970	1976	Projections				
					1980	1990	2000	2010	2020
65 years and over	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0
65 to 69 years	40.7	37.7	35.0	36.1	34.9	33.6	28.9	33.4	35.4
70 to 74 years	27.8	28.6	27.2	25.8	27.3	26.1	25.9	23.4	26.9
75 to 79 years	17.4	18.5	19.2	17.7	17.3	18.4	20.1	17.0	16.8
80 to 84 years	9.3	9.6	11.5	11.9	11.3	12.2	13.3	13.1	10.2
85 years and over	4.8	5.6	7.1	8.6	9.2	9.7	11.8	13.1	10.6

Source: U.S. Bureau of the Census, "Current Population Reports," series P-25, Nos. 311, 519, 614, 643, and 704.

Table IV–4.

Familial aged dependency ratios and societal aged dependency ratios: 1920 to 2020

[Figures are shown for July 1 of the year indicated. Ratios for 1940 and later years include Armed Forces overseas.]

Year		Population 65 to 79 years Population 45 to 49 years	Population 65 years and over Population 18 to 64 years
Estimates:			
1920		0. 76	. 08
1930		0. 82	. 09
1940		0. 95	. 11
1950		1. 16	. 13
1960		1. 29	. 17
1970		1. 35	. 17
1976		1. 56	. 18
Projections:			
1980		1. 80	. 18
1990		1. 68	. 20
2000	II	1. 25	. 20
Range	I		. 20
	III		. 20
2010	II	1. 27	. 20
Range	I		. 19
	III		. 21
2020	II	2. 16	. 26
Range	I		. 23
	III		. 29

Source: U.S. Bureau of the Census, Census of Population, 1930, *General Report*, vol. II, table 7, and "Current Population Reports," series P–25, nos. 311, 519, 614, 643, and 704; unpublished data for age group 45–49 for years 2010 and 2020.

live with their children. The trend in the relative numbers of persons 65 to 79 and persons 45 to 49 can be used to illustrate the shifts in the ratio of elderly parents to children. This measure increased sharply and steadily between 1920 and 1976, when there were 156 persons 65 to 79 per 100 persons 45 to 49. A peak will be reached in 1980 at 180, and again in the year 2020 with a still higher peak of 216.

The fluctuations in these familial dependency ratios reflect mainly past trends in the number of births, which have tended to be wavelike in the last several decades. For example, the very high ratio in the year 2020 results from the combination of the relatively large (average annual) number of births in the postwar decade (population 65 to 74 years) and the small (average annual) number of births in the early seventies (population 45 to 49 years).

The shift in age structure is only one factor affecting the problem of the social, economic, and psychological support of the elderly. The prospective shifts in the age structure suggest that the extent of the problem of familial and societal support of the aged will fluctuate in the future but that, in general, it will tend to become greater than at present and at times serious.

Sex Composition

We look briefly next at the balance of the sexes among the older population. There were 69 males for every 100 females in the age group 65 and over in the United States in 1976 (table IV–5); that is, about 60 percent of the elderly population is female. This ratio is expected to fall to 67 per 100 by the year 2000 and then to rise, reaching 69 again by the year 2020. These figures are consistent with a sex ratio at birth of about 105. The deficit of males increases sharply with advancing age. The sex ratio at ages 75 and over, for example, is substantially lower than at ages 65 and over. It was 58 in 1976 and is expected to fall to about 54 by the year 2000; then it may remain at this level until 2020.

The main reason for the drop in the sex ratio *with increasing age* is that male mortality has been higher than female mortality at each age of life for many decades in the United States. The main factor determining the trend in the sex ratios at each age *over time* is the relative level of male and female mortality, or more precisely, the relative levels of male and female survival rates. The death rates for the sexes at the various age groups have been steadily diverging, to the greater and greater advantage of women. The further decline or slight rise in the sex ratio of the population at the older ages projected for future years is a result of the assumption made in preparing the projections that there will be a further divergence of male and female mortality rates (*4*). An historical analysis of mortality trends in the United States and comparative international analysis of mortality rates suggest that there will be only moderate reductions in future death rates and no substantial convergence of the death rates of the two sexes (*5*).

The large excess of females over males among the elderly is also reflected in the notable differences in the age distribution of the sexes. The percentages of the elderly in the total population of each sex were 12.3 for females and only 9.0 for males in 1976 (table IV–2), as compared with 10.7 percent for both sexes combined. In the year 2000, the percentage for elderly females may be as high as 15, compared with 11 for elderly males. For the age group 75 years and over, the difference in the percents for males and females is even more pronounced.

Geographic Distribution

According to the 1970 census data which classified the population by urban-rural and city-size categories, there are very large geographic variations in the relative numbers of aged in the population (table IV–6). The largest proportion (13.6) of the aged lived in rural-nonfarm areas

Table IV-5.

Sex ratios of the population, by broad age groups: 1950 to 2020

[Males per 100 females. Estimates and projections as of July 1. Figures include Armed Forces overseas.]

Age and projection series	1950	1960	1970	1976	Projections				
					1980	1990	2000	2010	2020
All ages II	99. 3	97. 0	94. 8	94. 8	95. 0	94. 8	94. 6	94. 4	93. 8
Range I					95. 1	95. 2	95. 4	95. 7	95. 7
Range III					94. 9	94. 5	94. 0	93. 5	92. 4
Under 15 years	103. 8	103. 4	103. 9	104. 2	104. 5	105. 1	105. 2	105. 1	105. 1
15 to 29 years	98. 7	97. 7	97. 8	100. 3	101. 3	101. 3	102. 1	102. 1	102. 1
30 to 44 years	97. 4	95. 5	95. 2	95. 8	96. 6	97. 2	96. 7	96. 8	97. 3
45 to 59 years	99. 8	96. 9	93. 4	94. 8	95. 3	95. 1	95. 9	95. 4	95. 3
60 to 64 years	100. 4	91. 2	87. 7	87. 9	87. 9	98. 8	89. 9	91. 1	90. 9
65 to 69 years	94. 0	87. 8	80. 7	79. 3	79. 7	80. 5	82. 4	82. 4	83. 2
70 to 74 years	91. 3	85. 3	73. 9	73. 5	72. 4	72. 9	74. 6	74. 8	75. 9
75 to 84 years	85. 0	77. 4	65. 9	61. 0	60. 3	59. 6	59. 9	60. 7	61. 4
85 years and over	70. 0	63. 8	53. 2	47. 0	44. 7	40. 9	39. 4	38. 8	38. 5
65 years and over	89. 5	82. 6	72. 0	69. 0	68. 2	67. 3	66. 6	67. 0	69. 3
75 years and over	82. 6	75. 0	63. 3	57. 7	56. 2	54. 7	54. 0	53. 4	54. 2

Source: U.S. Bureau of the Census, "Current Population Reports," series P-25, nos. 311, 519, 614, 643, and 704.

Table IV–6

Distribution of the total, white, black, and Spanish-heritage populations 65 years old and over by urban and rural residence and by size of place: 1970.

Race	1970										1960		
	Total	Urban						Rural			Total	Urban	Rural
		Total	Urbanized areas			Other places of—		Total	Places of 1,000 to 2,500	Other rural			
			Total	Central cities	Urban fringe	10,000 or more	2,500 to 10,000						
Number (thousands):													
Total	20,066	14,631	11,106	6,842	4,264	1,788	1,737	5,434	903	4,532	16,560	11,526	5,033
White	18,330	13,309	10,049	5,950	4,100	1,641	1,619	5,021	852	4,169	15,304	10,672	4,632
Negro and other races	1,735	1,322	1,056	892	164	147	118	413	51	362	1,256	854	402
Black	1,559	1,192	949	813	137	136	107	367	44	323	NA	NA	NA
Spanish heritage [1]	382	330	271	194	77	29	30	52	NA	NA	NA	NA	NA
Percent of all ages:													
Total	9.9	9.8	9.4	10.7	7.8	10.8	12.2	10.1	13.6	9.6	9.2	9.2	9.3
White	10.3	10.3	10.0	12.0	8.0	11.1	12.5	10.3	13.9	9.7	9.6	9.7	9.6
Negro and other races	6.8	6.4	6.0	6.2	5.3	8.3	9.3	8.4	9.9	8.2	6.1	5.8	7.1
Black	6.9	6.5	6.0	6.2	5.4	8.7	9.7	8.7	10.4	8.1	NA	NA	NA
Spanish heritage [1]	4.1	4.0	2.9	4.2	2.4	4.3	5.0	4.6	NA	NA	NA	NA	NA
Percent of all areas:													
Total	100.0	72.9	55.3	34.1	21.2	8.9	8.7	27.1	4.5	22.6	100.0	69.6	30.4
White	100.0	72.6	54.8	32.5	22.4	9.0	8.8	27.4	4.6	22.7	100.0	69.7	30.3
Negro and other races	100.0	76.2	60.9	51.4	9.5	8.5	6.8	23.8	2.9	20.9	100.0	68.0	32.0
Black	100.0	76.5	60.9	52.1	8.8	8.7	6.9	23.5	2.8	20.7	NA	NA	NA
Spanish heritage [1]	100.0	86.3	70.9	50.8	20.2	7.7	7.7	13.7	NA	NA	NA	NA	NA

NA = Not available.

[1] For New York, New Jersey, and Pennsylvania, persons of Puerto Rican birth and parentage only; for five southwestern States, persons of Spanish language or Spanish surname; for remaining States, persons of Spanish language. Note that persons of Spanish origin may be of any race.

Source: U.S. Bureau of the Census, "Census of Population:" 1970, "General Population Characteristics," Final Report, PC(1)-B1, United States Summary, table 52, and PC(1)-C1, United States Summary, table 118.

(places of 1,000 to 2,500). The high percentage of aged persons in such places may have resulted from a high rate of out-migration of young people to larger places and the in-migration of farmers over 65 years old from the nearby farming areas when they can no longer operate their farms. The latter factor and the high birth rate of the farm population may account for the much lower proportion of elderly persons on farms. In general, the larger the place, the lower the percentage of elderly people, with the urban fringe of urbanized areas showing the lowest percentage, 7.8 percent.

Of the 20.1 million persons 65 and over in April 1970, about one-third (34 percent) lived in central cities of urbanized areas and about one-quarter (27 percent) lived in rural areas. The black elderly were much more heavily concentrated in central cities than the white elderly. Of the 1.6 million blacks 65 and over in 1970, over half (52 percent) lived in a central city and about one-quarter (24 percent) lived in rural areas, mostly on farms. The population of Spanish heritage is also heavily concentrated in central cities (51 percent), but an important share also lives in the urban fringe (20 percent).

Migration rates for the elderly are relatively low; with increasing age people migrate less. They often remain in very small towns, the rural hinterland, or large urban centers, particularly the deteriorated parts of these areas.

Many of the States in the midwestern farm belt (i.e., Iowa, Kansas, Missouri, Nebraska, and South Dakota), as well as Florida and Arkansas, showed high proportions (i.e., 12.5 percent or more) of elderly persons in 1976, while several States in the South (South Carolina and Maryland) and West (New Mexico, Utah, Nevada, Colorado), and the outlying States of Alaska and Hawaii showed low proportions (8.5 percent or less) of elderly persons (See figure IV–2). High proportions tend to result from continued out-migration of young persons, substantial in-migration of older persons in recent years, heavy immigration of foreign-born persons, particularly prior to 1925, and relatively low fertility, while low proportions tend to result under the opposite conditions. In about one-fifth of the counties in the West North Central Division of the United States, over 20 percent of the population in 1976 was over 65 years of age.

Mortality and Survival

From 1954 until 1968 there was little progress in the reduction of mortality in terms of age, sex, race, and cause-of-death categories. The expectation of life at birth and at age 65 remained nearly the same over the 10-year period from 1955 to 1965 (table IV–7). Since 1968 the mortality of the older population has begun to fall and the average rate of decline was greater at each age group 55 and over than during 1940–54, the earlier period of rapid mortality decline (table IV–8). In the 9-year period from 1968 to 1977, for example, there was an 18 percent decline in the death rate for those 65 to 74, as compared with a 22 percent de-

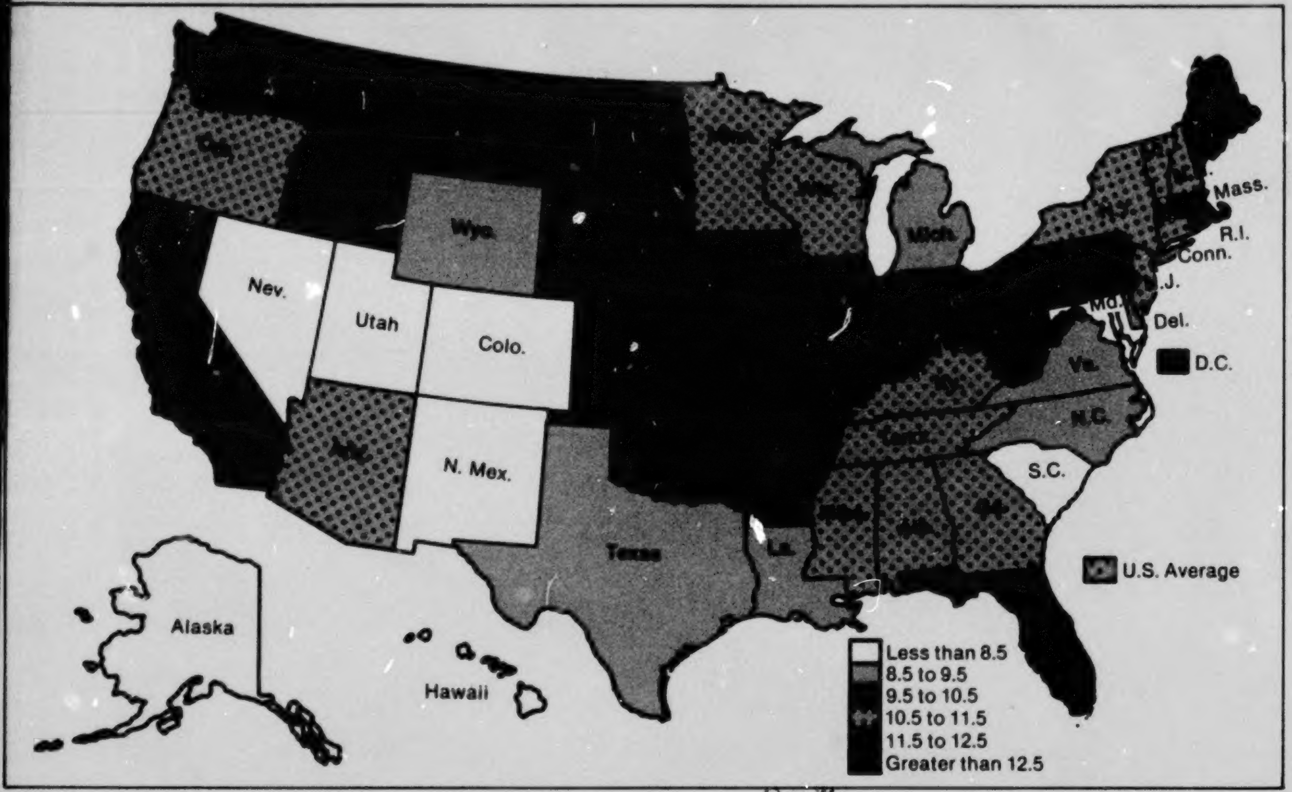
Wyo.
Nev.
Utah
Colo.
N. Mex.
Texas
La.
Mich.
Mass.
R.I.
Conn.
N.J.
Md.
Del.
D.C.
Va.
N.C.
S.C.
U.S. Average
Alaska
Hawaii
Less than 8.5
8.5 to 9.5
9.5 to 10.5
10.5 to 11.5
11.5 to 12.5
Greater than 12.5

Figure IV–2.

Percent 65 years old and over of the total population for States: 1976.

Source: Bureau of the Census, unpublished data.

cline for this age group in the 14-year period from 1940 to 1954. The observed 14 percent average decline for the combined ages 65 and over during the recent period understates the real decline for the component ages because of the aging of the elderly population itself.

Sex Differences.—We have already commented on the sex difference in mortality. This difference is quite large and has been growing until recently. By 1976, the difference in the life expectancy at birth of males and females reached 7.7 years. It was only 4.5 years in 1940. An increasing part of the difference is accounted for by the difference in the life expectancy at age 65. According to the death rates of 1976, the male-female difference in the average number of years lived in the age interval 0 to 64 years was 2.3 years, as compared with 4.3 years for ages 65 and over (table IV–7). The death rate for males 65 and over exceeded the corresponding rate for females by 46 percent in 1976 (table IV–9). In 1940, the difference was less than half as great.

The large sex differences in mortality have considerable consequences for many aspects of our social life. For example, they tend to result in a high rate of widowhood and paternal orphanhood, large excesses of women at the older ages, major economic losses to families and society, and loss of familial psychological and social support for the surviving women.

The gap between male and female death rates and longevity and the basis for the gap are matters of serious concern, therefore. Quite apart from the evident desirability of reducing mortality where it is excessively high, the gap is evidence of a considerable inequality between the sexes in the effect of environmental and/or genetic influences on mortality rates.

The evidence for the basis of the large and growing difference is conflicting, as suggested by a number of facts and studies. Studies by Enterline (*6*), Godley and Kruegel (7), Retherford (*8*), and Waldron (*9*) favor explanations primarily in terms of "environmental" factors. Others, such as the Madigan study (*10*), favor explanations in terms of genetic and nongenetic hereditary factors. The study of Madigan, relating to mortality differences between brothers and nuns in Catholic teaching orders, who presumably had essentially the same life-styles, shows an excess of male mortality. Male fetal and infant mortality is markedly greater than female fetal and infant mortality. The lengthening of the female reproductive period over the generations, presumably due to improved nutrition and hygiene, has apparently afforded an increasing biological advantage to women.

Many of the changes over time in the difference between male and female death rates appear to be associated with social and environmental factors. The Retherford study concluded that two-thirds of the divergence between the male and female mortality rates of adults in most of this century was due to smoking. Yet, females have taken up smoking in large and increasing numbers since World War II. Furthermore, during the same period women have been entering the labor force in large and increasing numbers. We should have seen a major effect

Table IV-7.

Life expectancy at birth and at age 65, by race and sex: 1900 to 1976

Race and year	At birth				At age 65			
	Both sexes	Male	Female	Difference	Both sexes	Male	Female	Difference
All races:								
1900–02	49.2	47.9	50.7	2.8	11.9	11.5	12.2	0.7
1939–41	63.6	61.6	65.9	4.3	12.8	12.1	13.6	1.5
1954	69.6	66.7	72.7	6.0	14.4	13.1	15.7	2.6
1968	70.2	66.6	74.0	7.4	14.6	12.8	16.3	3.5
1976	72.8	69.0	76.7	7.7	16.0	13.7	18.0	4.3
White:								
1900–02	49.7	48.2	51.1	2.9	11.9	11.5	12.2	0.7
1939–41	64.9	62.8	67.3	4.5	12.8	12.1	13.6	1.5
1954	70.5	67.4	73.6	6.2	14.4	13.1	15.7	2.6
1968	71.1	67.5	74.9	7.4	14.7	12.8	16.4	3.6
1976	73.5	69.7	77.3	7.6	16.1	13.7	18.1	4.4
Black and other races:								
1900–02	33.8	32.5	35.0	2.5	10.9	10.4	11.4	1.0
1939–41	53.9	52.3	55.6	3.3	13.0	12.2	13.9	1.7
1954	63.4	61.0	65.8	4.8	14.6	13.5	15.7	2.2
1968	63.7	60.1	67.5	7.4	13.7	12.1	15.1	3.0
1976	68.3	64.1	72.6	8.5	15.8	13.8	17.6	3.8
White-black differences:								
1900–02	15.9	15.7	16.1	(1)	1.0	1.1	0.8	(1)
1939–41	11.0	10.5	11.7	(1)	−0.2	−0.1	−0.3	(1)
1954	7.1	6.4	7.8	(1)	−0.2	−0.4	—	(1)
1968	7.4	7.4	7.4	(1)	1.0	0.7	1.3	(1)
1976	5.2	5.6	4.7	(1)	0.3	−0.1	0.5	(1)

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[1] Not applicable.

Source: National Center for Health Statistics (U.S. Public Health Service) and the U.S. Bureau of the Census. For 1976, "Advance Report—Final Mortality Statistics, 1976," "Monthly Vital Statistics Report," Vol. 26, No. 12, supplement (2), March 1978.

Table IV-8.

Death rates for the population 55 years old and over, by age: 1940 to 1977

Year and period	55 to 64 years	65 to 74 years	75 to 84 years	85 years and over	65 years and over
Rates per 1,000 population:					
1940	22.2	48.4	112.0	235.7	72.2
1954	17.4	37.9	86.0	181.6	58.6
1968 [1]	17.0	37.2	82.9	195.8	61.4
1976 (prov.)	14.8	31.4	73.2	156.1	54.2
1976	14.8	31.3	73.3	155.7	54.3
1977 (prov.)	14.3	30.5	71.5	145.9	52.6
Percent change:					
1940–54	−21.6	−21.7	−23.2	−23.0	−18.8
1954–68	−2.3	−1.8	−3.6	+7.8	+4.8
1968–77	−15.9	−18.0	−13.8	−25.5	−14.3

[1] Official estimates for 1968 have been revised by the Census Bureau on the basis of revised population estimates.

Source: National Center for Health Statistics (U.S. Public Health Service), various annual volumes of "Vital Statistics of the United States" and various issues of "Monthly Vital Statistics Report."

Table IV-9.

Ratios of male to female death rates for the population 55 years and over, by age: 1900 to 1976

Year	55 to 64 years	65 to 74 years	75 to 84 years	85 years and over	65 years and over
1900	1.14	1.11	1.08	1.05	1.06
1940	1.45	1.29	1.17	1.08	1.17
1954	1.82	1.57	1.29	1.06	1.30
1968	2.07	1.88	1.46	1.18	1.44
1976	1.99	1.97	1.58	1.26	1.46

Source: U.S. Bureau of the Census, "United States Life Tables, 1930," 1936; National Center for Health Statistics (U.S. Public Health Service), annual volumes of "Vital Statistics of the United States," for 1940 and 1954, and "Advance Report—Final Mortality Statistics," "Monthly Vital Statistics Report," vol. 26, No. 12, supplement (2), March 1978.

on the death rates of women and some convergence in the rates. In other words, the divergence of male and female death rates has been occurring in spite of the fact that the differences in the life-styles and roles of men and women have diminished. It is notable that we find a difference in life expectancy of some 10 years in the USSR (*11*), where men and women occupy much more equal occupational roles than in the United States. Clearly social factors exercise an important influence on the difference, but in combination with and in interaction with genetic and biological factors.

The increasing male-female difference may also be interpreted as a result of a major shift of the cause-pattern of mortality in this century, involving a sharp reduction in the relative importance of the infectious and parasitic illnesses and maternal mortality and a rise in the relative importance of the "chronic degenerative" diseases, e.g., diseases of the heart, malignant neoplasms, cerebrovascular diseases. For the first group of causes, female rates tend to be higher than or equal to those of male rates, while for the second group of causes, male rates tend to be much higher. Even for the latter group, male-female differences have widened over the last several decades.

Race Differences.—There is a difference of several years in life expectancy at birth between the races (white, black-and-other races). The difference has been reduced sharply over the last several decades, from about 11 years in 1940 to only 5 years in 1976 (table IV–7). The difference in life expectancy at age 65 is small, however, and has been small for decades. Improvements in black mortality have been much greater than in white mortality at ages under 65, but there has been little change in the difference at ages above 65.

Death rates for the black-and-other-races population are higher than for whites below age 65 and at ages 65–74. There is then a crossover of the rates at ages 75–79 in the U.S. death registration data. The crossover is not to be viewed merely as a statistical artifact, since the crossover phenomenon appears in many other paired comparisons, often involving data of high quality (*12*). For example, it appears in death rates based on U.S. Social Security Administration data, albeit at a higher age (ages 80 to 84). The Social Security data also reflect smaller differences between the races than the registration statistics.

Little attention has been given to the relative contribution of genetic and environmental influences to the white-black mortality gap. The Kitagawa and Hauser study suggests that much of it may be accounted for by differences in the socioeconomic status of the racial groups (*13*).

Causes of death.—The cause-pattern for deaths of the elderly continues to shift toward the chronic (endogenous) causes and away from the infectious-parasitic causes. The major endogenous causes are moving in opposite directions, however; death rates from cancer have been rising and death rates from cardiovascular diseases have been falling.

Only a few categories of causes account for most deaths over age 65. Diseases of the heart account for 44 percent of all deaths at these ages, and diseases of the heart, malignant neoplasms, and cerebrovascular diseases account for three-quarters of the deaths (table IV–10). The rates for the remaining "leading" causes are so low that each cause, taken separately, accounts for less than 5 percent of the total number of deaths over age 65.

Additional insight into the cause-pattern of mortality is secured by examining the possible gain in life expectancy from eliminating various causes of death and the chance of eventually dying from various causes. If all deaths from cardiovascular diseases were eliminated, the gain in life expectancy at birth would be 12 years and the gain at ages 65 would be 11 years (table IV–11). Diseases of the heart account for about half of these additions. Roughly speaking, a 65-year-old has a two-thirds chance of dying from a cardiovascular disease—a 50 percent chance of dying from a disease of the heart and a one-seventh chance of dying from a cerebrovascular disease. If all cancer mortality were eliminated, 2½ years of life would be added at birth and 1½ years of life at age 65. The chance of eventually dying from cancer is one-seventh at age 65. The elimination of cancer would have a much smaller relative effect on life expectancy at age 65 than the elimination of cardiovascular diseases because cancer occurs more evenly over the age distribution than the cardiovascular diseases.

Table IV–10.

Death rates for the 10 leading causes of death for ages 65 and over, by age: 1976

[Deaths per 100,000 population]

Cause of death by rank	65 years and over	65 to 74 years	75 to 84 years	85 years and over
All causes	5,428.9	3,127.6	7,331.6	15,486.9
1. Diseases of the heart	2,393.5	1,286.9	3,263.7	7,384.3
2. Malignant neoplasms	979.0	786.3	1,248.6	1,441.5
3. Cerebrovascular diseases	694.6	280.1	1,014.0	2,586.8
4. Influenza and pneumonia	211.1	70.1	289.3	959.2
5. Arteriosclerosis	122.2	25.8	152.5	714.3
6. Diabetes mellitus	108.1	70.0	155.8	219.2
7. Accidents	104.5	62.2	134.5	306.7
Motor vehicle	25.2	21.7	32.3	26.0
All other	79.3	40.4	102.2	280.7
8. Bronchitis, emphysema and asthma	76.8	60.7	101.4	108.5
9. Cirrhosis of liver	36.5	42.6	29.3	18.0
10. Nephritis and nephrosis	25.0	15.2	34.1	64.6
All other causes	677.5	427.8	908.6	1,683.8

Source: National Center for Health Statistics (U.S. Public Health Service), "Advance Report—Final Mortality Statistics, 1976," Monthly Vital Statistics Report, vol. 26., No. 12, supplement (2), March 1978; and unpublished data provided by the National Center for Health Statistics.

Table IV–11.

Gain in life expectancy from eliminating specified causes of death and chance of eventually dying from these causes: 1969–1971

Cause of death	Gain in life expectancy (in years)		Chance of eventually dying	
	At birth	At age 65	At birth	At age 65
Major cardiovascular—renal diseases	11.8	11.4	.588	.672
Diseases of the heart	5.9	5.1	.412	.460
Cerebrovascular diseases	1.2	1.2	.122	.149
Malignant neoplasms [1]	2.5	1.4	.163	.145
Motor vehicle accidents	.7	.1	.020	.006
All other accidents	.6	.1	.026	.018
Influenza and pneumonia	.5	.2	.034	.037
Diabetes mellitus	.2	.2	.020	.021
Infective and parasitic diseases	.2	.1	.007	.005

[1] Including neoplasms of lymphatic and hematopoietic tissues.

Source: National Center for Health Statistics, (U.S. Public Health Service), "U.S. Life Tables by Cause of Death: 1969–71," by T. N. E. Greville, "U.S. Decennial Life Tables for 1969–71," vol. 1, No. 5, 1976.

If deaths due to cardiovascular diseases were eliminated, the size of the total population and the number of elderly people would increase tremendously *(14)*. The proportion of those aged 65 and over would increase sharply in the first few decades, and then would continue to increase more slowly for several decades. The increase in the percent of the elderly resulting from the elimination of cancer would be far less than the increase resulting from the elimination of cardiovascular diseases. Elimination of any important cause would tend to raise the sex ratio of the elderly. For example, the present sex ratio of 69 males to 100 females might rise by several points in a single decade if cardiovascular diseases were eliminated.

It should be clearly recognized that these figures have no direct implications for the future since complete elimination of any category of the cardiovascular diseases or of cancer has not been projected or predicted. They simply elucidate the current cause-pattern and have implications for planning current health programs most effectively.

Prospects for Mortality Reduction.—A number of different approaches may be taken in projecting death rates for the United States. One is to extrapolate past trends in age-sex-race-specific death rates. Another is to consider the rates in more analytic terms, for example, in terms of causes of death and, even further, in terms of the factors affecting specific causes of death. An even more analytical step is to take account of competing risks and joint causes of death. Guides for extrapolation may be derived from the experience of countries with the lowest rates and from available studies of the biological "limit" of mortality.

The Census Bureau's latest projections of mortality, incorporated in its latest projections of population, are adaptations of mortality projections prepared by the Social Security Administration that took explicit account of past trends in death rates for age, sex, and cause categories. They imply expectations of life at birth and at age 65 in 2020 only a little greater than at present. For the year 2020, an average life expectancy of 75.0 years was projected for both sexes, as compared with the base estimate of 73.1 years in 1976 (table IV-12). For males separately, the life expectancy value set for 2020 is 70.7 years, as compared with 69.1 years for 1976. The corresponding figures for females are 79.4 and 77.0. These figures imply a modest continuation of the divergence between male and female death rates and life expectancy that has been observed so far in this century.

It is of value in understanding the possible influence of future changes in mortality on population size and structure to posit certain highly hypothetical conditions of mortality, namely the immediate or gradual elimination of all deaths. Under an assumption of "immediate immortality" (with series II fertility), 38 percent of the population in the year 2025 would be over 65, and under an assumption of "gradual immortality," some 33 percent of the population would be over 65 in that year (*15*). The phrase "gradual immortality" is used here to mean approximately that death rates were set at zero in the year 2000 and that death rates for intermediate years were obtained by geometric interpolation. The purpose of such an exercise is not to make predictions; it is to focus on a possible trajectory of mortality and population change.

Uniform percentage reductions in death rates, such as are implied in this exercise, would tend to result in much greater percentage increases in survival rates, and hence in population numbers, at the older ages than at the younger ages. As a result, the proportion of the elderly would rise sharply and continue to grow rapidly. The rise in the proportion would be reinforced by declining fertility if the latter were to occur. The effect of a concentration of future (absolute) declines in death rates and future (relative) increases in survival rates at the older ages—a virtual certainty in the light of the present age structure of mortality if large reductions occur in mortality at all—is elucidated by showing the effect of extreme uniform changes in mortality on population size and structure.

Historical and comparative international analysis suggest only moderate reductions in death rates in the United States in the next several decades, however (*16*). There is little reason to anticipate major increases in life expectation in this period and it is visionary to anticipate any extension of human life span (*17*). Life expectation at birth in Sweden in 1974 and for a composite "country" derived from the lowest death rates at each age for various countries around 1974 is only 3 to 5 years greater than for the United States in 1974 (table IV-12).

Comparative international and historical demographic analysis also suggests no great convergence of male and female mortality or life

Table IV-12.

Recent and projected values for life expectancy at birth and at age 65 for the United States and comparative international values

Year	At birth				At age 65			
	Both sexes	Male	Female	Difference [1]	Both sexes	Male	Female	Difference [1]
1973 [2]	71.3	67.6	75.3	7.7	15.3	13.1	17.2	4.1
1974 [3]	71.9	68.2	75.9	7.7	15.6	13.4	17.5	4.1
1975 [4]	72.5	68.7	76.5	7.8	16.0	13.7	18.0	4.3
1976 [5]	72.8	69.0	76.7	7.7	16.0	13.7	18.0	4.3
Projections: [6]								
1976 (base)	73.1	69.1	77.0	7.9	16.0	13.8	18.4	4.6
2000	74.1	70.0	78.3	8.3	16.8	14.2	19.0	4.8
2020	75.0	70.7	79.4	8.7	17.2	14.5	19.8	5.3
2050	76.4	71.8	81.0	9.2	18.0	15.0	20.7	5.7
Best country composite, 1974	76.6	73.8	79.4	5.6	17.4	15.5	18.8	3.3
Sweden, 1974	75.1	72.2	78.0	5.8	16.0	14.1	17.5	3.4
Difference, United States and best country composite, 1974	−4.7	−5.6	−3.5	2.1	−1.8	−2.1	−1.3	0.8

[1] Excess of female over male value.

[2] National Center for Health Statistics, "Final Mortality Statistics, 1973," "Monthly Vital Statistics Report," vol. 23, no. 11, supplement (2), February 1975.

[3] National Center for Health Statistics, "Final Mortality Statistics, 1974," "Monthly Vital Statistics Report," vol. 24, no. 11, supplement, February 1976.

[4] National Center for Health Statistics, "Final Mortality Statistics, 1975," "Monthly Vital Statistics Report," vol. 25, no. 11, supplement, February 11, 1977.

[5] National Center for Health Statistics, "Final Mortality Statistics, 1976," "Monthly Vital Statistics Report," vol. 26, no. 12, supplement (2), March 30, 1978.

[6] U.S. Bureau of the Census, "Projections of the Population of the United States: 1977 to 2050," "Current Population Reports," series P-25, no. 704, July 1977, and unpublished data.

expectancy in the United States in the near future. When we compare the U.S. values for 1974 with the figures for Sweden in 1974 and the country composite, we see that the U.S. figures are much closer to the attained "best" levels for females than for males. The comparison suggests that the possibilities for improvement for males in the United States are greater than for females and that some convergence in the values for males and females may be realized. This analysis also suggests that the male-female difference may have a substantial, irreducible minimum in post-industrial society. The present large difference presumably reflects both a biological advantage of women and the persistent, sizeable differentiation of the life-styles and roles of the sexes. Even if men are given equal environmental "opportunities" and, further, environmental "preferences" over women, a large part of the current difference is expected to remain for several decades.

Some Implications for Health Care

Having summarized the facts regarding the principal prospective demographic changes, I want to note some social and economic implications of these changes, particularly those relating to the current and future requirements for health services.

First, the demographic changes identified will mean that the needs of the elderly will account for a larger and larger share of the health effort, resources, and budget. The aged are the largest users of health resources. They make more visits to physicians (per person) and use hospital facilities more frequently than any younger age group. Given the great increase in the number of elderly expected in the next half century and even the same per capita demand for health care for each age-sex group in the future as at present, the total demand for health care will tend to increase greatly. We have noted also the expected rise in the proportion of elderly persons, reflecting the relatively more rapid increase in the number of elderly than of younger age groups, and the upward shift in the average age of the elderly. Demand for health care rises with age within the older age span. These population shifts will augment greatly the relative demand for health care on the part of the elderly in future years.

Next, health resources will have to be increasingly geared toward the health needs of older women. We have noted that the proportion of females among the elderly, which is already quite high, is expected to continue to rise, at least in the next few decades.

We can anticipate increasing utilization of health care resources per capita among the elderly because of the expected increase in the educational and income level of the elderly and the increased participation in broad health insurance plans. These changes will tend to lead the prospective patient to seek more comprehensive health care services and advanced methods of treatment and to resort to specialists and use of specialized equipment. At the same time, the past inequality in the use of health care services among various income classes, especially the relative disenfranchisement of the middle income group, has been changed by the advent of Medicare and private health insurance programs.

In recent years the population in nonmetropolitan areas has been growing more rapidly than the population in metropolitan areas. A serious imbalance already exists between the distribution of the elderly population and the distribution of health resources in these areas. There is the problem of avoiding further inbalances resulting from further population redistribution. The problem of the availability of health personnel applies also to the inner zones of the city where there is a differential concentration of poor elderly persons.

Some extended implications of the prospective demographic changes affecting the older population, suggesting areas of research and action, are as follows:

(1) The ratio of the number of elderly persons to the number of younger persons of the next generation will become less favorable in the long run. With declining or low fertility, elderly persons will have fewer brothers and sisters and fewer children than in the past to provide needed or desirable economic, social, and psychological support. The wide gap between male and female mortality rates and the tendency toward "nucleation" of American families and "individuation" of American households assure that most elderly persons will be female, be widowed, and live alone.

More studies are needed relating to (a) the kinship network of elderly persons and to the social, economic, and psychological support that relatives and friends give to elderly people, (b) the degree of contacts and/or isolation of elderly persons, and (c) the effect of migration on the family structure and kinship network/social contacts of older persons. Given the relation of isolation to emotional disorders and the prominence of emotional disorders among elderly persons, can a logical nexus be traced between the small family system (low fertility and nucleation of families and individuation of households), a large male-female mortality gap, and high internal migration rates of younger relatives, on the one hand, and the prevalence of emotional disorders among the elderly, on the other? Of course, any such relation is reinforced by low income, loss of the work role, poor physical health, and limited ambulatory ability on the part of many elderly persons.

(2) If, as is quite possible, the U.S. population becomes stationary in the next 50 years as a result of recent and prospective low fertility, the proportion of elderly persons who have living relatives will be much smaller while the proportion of elderly persons in the population will be much larger. It would be desirable to explore rather fully the implications of a stationary or nearly stationary population for the conditions of the elderly, including particularly the problem of the provision of health services. It is anticipated, for example, that the higher prospective ratio of aged persons to persons of the usual working ages (societal dependency ratio) and the smaller kinship network (familial dependency ratio) will necessitate a greater role of government in the support of the elderly, particularly in providing health and other services to the elderly (*18*).

(3) Studies showing the effects of migration of older persons on their health and longevity are needed. Some questions to be investigated are as follows: How does life expectancy at birth and at age 65 for persons born in the State of residence differ from life expectancy for persons living in the State but born in another State? Is life expectancy for in-migrants to certain States different from that for those who have lived in these States all their lives? We need to ascertain the relative contribution of "early life imprints" in the State of birth and influences in the State of later residence. We still do not know what factors account for the small, but persistent, State variations in longevity, i.e., the basis of the advantage of the North Central States over the South and Northeast.

What happens to the morbidity and mortality rates of persons who move to retirement areas as compared with those who do not move (controlling, if possible, for selective factors relating to health)? Should society play an active role in encouraging people to retire to areas showing lower post-retirement morbidity and mortality rates?

(4) A special type of mobility in later life is movement from private households to "group quarters," such as nursing homes and rest homes. An estimated 5 percent of the population 65 and over currently reside in institutions but perhaps a much larger percent of elderly persons spend some substantial time in an institutional setting. An important area of concern is the effect on the physical and emotional health of elderly persons of moving to and residing in such group quarters as compared with remaining in their own private households or going to live with their children. We still do not know what living arrangements for the elderly will produce the lowest morbidity and mortality and the greatest measure of life satisfaction. Are we seeking to avoid institutionalization of elderly persons to enhance their physical and mental health, to save community funds, or to suit social convenience? How large a part does the current household status of the person play in the risk of institutionalization?

(5) In view of the considerable differences between the mortality levels of males and females and the tremendous demographic, social, and economic consequences that flow from these differences, it is important to study them intensively and to devote special attention to the relative etiological roles of genetic and environmental factors. More needs to be learned about those aspects of the behavior and genetic makeup of males and females which make the males more vulnerable than females to serious illness. Are there, for example, male-female differences in recognizing the need for, seeking, and accepting medical care? We should conduct the equivalent of KAP (knowledge-attitude-practice) studies for mortality, as is widely done for fertility in the less developed countries. Can the psychological, social, and physiological bases for the female advantage be more positively identified?

(6) Whether or not we can fully understand the underlying bases of the male/female mortality differential, its great magnitude, its persistence, and its many and profound consequences justify a major societal assault on the problem. Imaginative and wide-ranging programs have to

be launched with the prevention of premature death of males as a specific goal. Whether the following suggestions meet the needs can be argued. Nevertheless, I would venture that perhaps we need an Equal Health Opportunity (EHO) program which would aim at achieving near equality, if not complete equality, in the life expectancy of the two sexes by "affirmative action" (*cf.* EEO programs). The social goal of greater equality may be viewed as sufficiently important to call for a course of preferential treatment for men. A few, somewhat unrefined suggestive illustrations of appropriate steps may be mentioned: Give males preference in securing private medical appointments at preferred hours (e.g., at beginning and end of day), allow males higher deductions for medical expenses in their income tax returns (*cf.* extra exemptions for the aged and blind), allow males a higher sick-leave earning-rate at their jobs than women, offer all males over age 30 free employer-or community-sponsored medical examinations, etc.

(7) The same issues as presented in items five and six may be presented in relation to the black-white mortality differentials *mutatis mutandis*, although here the differences are not as marked and have been declining, the social consequences are not as profound, and the relative levels of the rates for the races shift at the older ages. The issues here relate primarily to the substantial excess of the mortality of blacks over whites at the ages below about age 75 as well as to the black advantage at the higher ages. Is it a tenable hypothesis that selective survival for a cohort following an early history of social and psychological deprivation contributes to lower death rates for the cohort at the older ages?

(8) In view of the pronounced socioeconomic differentials in mortality found in the 1960 Chicago mortality study (*19*), it would now appear fruitful to undertake a current study of national scope to determine whether the differentials have persisted and what aspects of socioeconomic status are now most closely related to longevity. Methodological alternatives to be considered include another census—death certificate match study, a follow-back study of a sample of death certificates, or an ecological study involving a sample of small geographic areas classified by socioeconomic status.

(9) In view of the trend toward declining proportions in the work force at the older ages, particularly of males, we should be concerned with the relation between work activity/retirement and health/mortality (allowing for the selective factor linking poor health and retirement).

(10) What do we know of the effects on the health of an older person of a loss of mate? Is there a relation between bereavement and deterioration in health, given the low remarriage rate of elderly widows?

(11) The sheer growth in the numbers of elderly persons and their special requirements for health services suggest the need for expanded training and accreditation of appropriate manpower in a number of health professions, i.e., geriatric social work, geriatric nursing, and geriatric medicine (especially herontic psychiatry).

(12) Various bioethical considerations are involved in decisions regarding the provision of health care to elderly people and intensive

studies of the issues are needed. In particular, there is a problem of making decisions about the allocation of limited resources. Available funds are always assigned on a discretionary basis to different public programs and, within the domain of health, to different programs for health prevention, maintenance, and treatment (*20*).

We have to resolve such issues as whether it is more useful to allocate funds to maintain the life of very sick, extreme-aged persons at all costs or to apply some of these funds for preventive purposes to younger adults. The latter course could, in the long run, be more effective in maintaining and improving the average quality of life among the elderly and reducing the inequality of male and female death rates. For example, would it not be more appropriate to spend a larger share of community funds on mobile cardiac units, use of which might prevent the death of many middle-aged husbands and fathers, and less on drastic mechanical methods of life-prolongation for extreme-aged persons who are already very seriously ill?

(13) As increasing numbers of persons survive to the older ages and particularly as the proportion of older persons in the population increase, the relative frequency of persons with chronic, seriously debilitating conditions of later life, including incurable and intractable conditions, will increase. This development suggests the desirability of pursuing intensive research into the complex bioethical issues relating to the "right to die" and suggests the need to explore the concepts of "life with dignity" and the "right to die" as basic human rights.

Notes and References

(*1*) For a more comprehensive treatment of this subject, see Siegel, J. S.: Demographic Aspects of Aging and the Older Population in the United States. U.S. Bureau of the Census. *Current Population Reports*, series P-23, No. 59, Washington, D.C., U.S. Govt. Print. Off., 1976 (Second Printing, rev. 1978).

(*2*) **U.S. Bureau of the Census:** Projections of the Population of the United States: 1977 to 2050. *Current Population Reports*, series P-25, No. 704, Washington, D.C., U.S. Govt. Print. Off., 1977. The projections given in the present paper differ from those found in the study of the Bureau of the Census cited in footnote 1, mainly because the present projections use lower projected mortality rates. As a result the population projections have been increased by about 4 percent for the year 2000 and by about 5 percent for the year 2020.

(*3*) The basic fertility assumptions corresponding to the three series of population projections relate to the average numbers of children born to a woman in her lifetime:

Series I	2.7 children per woman
Series II	2.1 children per woman
Series III	1.7 children per woman

Only one mortality assumption and one immigration assumption (400,000 net immigration per year) are combined with these three fertility assumptions in the population projections.

(*4*) The divergence assumed is not very great. The fact that a rise in the population sex ratio at the ages 65 and over occurs even though a further divergence in death rates has been assumed is due to the fact that the arithmetic effect depends more directly on the relative difference in the corresponding survival rates.

(5) See discussion below. *See also* **Bourgeois-Pichat, J.**: Future Outlook for Mortality Decline in the World. Presented at the meeting of the United Nations, Ad Hoc Group of Experts on Demographic Projections, New York, November 1977.

(6) **Enterline, P. E.**: Cause of death responsible for recent increases in sex mortality differentials in the United States. *Milbank Mem. Fund Q.* 39:312–338, 1961.

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(10) **Madigan, F. C.**: Are sex mortality differentials biologically caused? *Milbank Mem. Fund Q.* 35:203–223, 1957.

(11) **Vallin, J. and Chesnais, J.**: Évolution récente de la mortalité en Europe, dans les pays Anglo-Saxons et en Union Soviétique, 1960–1970, *Population* 29:862–898, 1974, especially p. 863; Institut National d'Études Démographiques: Sixième rapport sur la situation démographique de la France. *Population* 32:253–338, 1977, especially 293–294.

(12) **Nam, C. B. and Ockay, K. A.**: Factors Contributing to the Mortality Crossover Pattern: Effects of Developmental Level, Overall Mortality Level, and Causes of Death. Paper submitted to the XVIII General Conference of the International Union for the Scientific Study of Population, Mexico City, August 8–13, 1977.

(13) **Kitagawa, E. M. and Hauser, P. M.**: *Differential Mortality in the United States: A Study in Socioeconomic Epidemiology.* Harvard University Press, Cambridge, 1973, esp. Chapters 2 and 8.

(14) **Preston, S. H.**: *Mortality Patterns in National Populations.* New York, Academic Press, 1976, esp. Chapter 7.

(15) **Siegel, J. S.**: *Current Population Reports*, Series P-23, No. 59, *op. cit.*, esp. p. 44.

(16) *See* Bourgeois-Pichat, *op. cit.*, for a more comprehensive analysis.

(17) For a summary comment on the last point, *see* **Neugarten, B. L.**: The future and the young-old. In Jarvik, L. F. (ed.) *Aging into the 21st Century: Middle-Agers Today.* New York, Gardner Press, Inc., pp. 137–152, 1978, especially page 140.

(18) **Shanas, E. and Hauser, P. M.**: Zero population growth and the family life of old people. *J. Soc. Issues* 30:79–92, 1974.

(19) **Kitagawa and Hauser,** *op. cit.*

(20) **Fuchs, V. R.**: *Who Shall Live? Health, Economics, and Social Change.* New York, Basic Books, 1974, and Rhoades, S. E.: How Much Should We Spend to Save a Life? *Public Interest* 51:74–92, 1978.

Discussion

Waldron: You have mentioned the sex difference in fetal mortality, but the picture is not entirely consistent. For instance, in pairs of twins of opposite sex or triplets or higher order births, there is no sex difference in fetal mortality. This could indicate that the male fetus is not inherently less viable, but rather the uterine environment may be less favorable when a male fetus is present. The second thing I want to mention has to do with the Madigan study, which found that the nuns lived longer than the brothers. The brothers smoked and drank more than the nuns did, so the brothers were exposed to a greater risk behaviorally. I also wanted to comment on your remarks about the effects of employment. I do think you are right in saying that if employment has a big impact on mortality, then the effect of women's increasing employment should have shown up more strongly than it has. I am tempted to speculate that one of the crucial factors may turn out to be socialization for employment. For instance, type A behavior is more common in men than in women. I think that may be due to the way that boys are socialized as compared to the way that girls are socialized. I do not think that recent changes in women's employment have had a big effect yet on the way that children are socialized, and certainly those children who have been socialized differently are not old enough to be dying in any significant numbers yet.

Palmore: In addition to the article on smoking and drinking, Madigan came out with another article reporting how much stress the brothers suffered trying to live up to their vows of poverty, chastity, and obedience. The nuns did not seem to suffer as much.

Cohen: That is nice evidence for the point I was going to make. Look at the way that women cope with the stresses that they have to deal with. I think there is a whole body of information on how people make use of resources. Women have alternatives open to them in this society, e.g., using social support systems, that men do not. That may make a difference which we have not yet studied systematically.

Gruenberg: Listening to this discussion reminds me of the fact that almost all the great killers discriminate against the weak, the old, and the young, quite aside from sex. These groups die at higher rates from all the epidemics, diseases, and if injured, they are more likely to die.

Palmore: I am puzzled by one question. Are 75-year-olds different from 65-to 74-year-olds? Have you noticed in your data any natural cutting off points for step increases at 75 or 80? My impression is that it is all linear.

Siegel: If you graph the whole series, you will observe a smooth curve changing more and more rapidly. The ages 65 and 75 are cutoffs partly created by ourselves. Numbers ending in five, or age groups corresponding to them, are a convention—that is the way we think—and so the Census data are tabulated that way. If you do graph the cumulative values for 65 to 74, 75 to 84, and 85 and over, I think you will see the gradualness of those changes and their relatively smooth curvilinear form.

Morbidity and Health Care Utilization

Mary Grace Kovar

National Center for Health Statistics
3700 East-West Highway
Hyattsville, MD 20782

Epidemiology, generally regarded as the study of the distribution and determinants of disease in human populations, is a science well suited to study of utilization of medical services by specified population groups such as the aging. It is essentially a quantitative science concerned with natural populations. Clinical trials may demonstrate the effectiveness of new treatment in a research setting; the epidemiologist is needed to determine whether the treatment is successful in preventing or curing disease in the population. To do so he needs to know the characteristics of the population requiring treatment, the characteristics of those providing treatment, and the setting in which treatment is provided. Only then can he determine whether the treatment is actually effective in practice.

The epidemiologist cannot accomplish these tasks alone. Medical sociologists, health economists, psychologists, and specialists in many other disciplines are required if we are to evaluate the effectiveness of medical care in preventing or curing disease or in alleviating symptoms or distress.

The elderly or aged population, which I shall define here for statistical convenience as those age 65 or older, is at high risk of disease and at high risk of utilizing medical services. Only 11 percent of the U.S. population is in this age group. Yet 64 percent of all deaths occur to people 65 or older—75 percent of all deaths from heart disease, 59 percent of all deaths from malignant neoplasms, 84 percent of all deaths from cerebrovascular disease. About 5 percent of the people age 65 or older die each year (1).

At any given time over 5 percent of the aged are in nursing homes or other facilities for long-term care. Among those living in the community the reported level of chronic conditions is high; arthritis, 38 percent; hearing impairments, 29 percent; vision impairments, 20 percent; heart conditions, 20 percent; and hypertension without heart involvement, 20 percent.

The very high levels of utilization of conventional health services—visits to physicians, inpatient care in general hospitals, and care in nursing homes—have been well documented. People age 65 or older account for 15 percent of all visits to office-based physicians, 34 percent of all days utilized in short-stay hospitals, and 89 percent of all residents of nursing homes (2).

They account for 29 percent of all monies spent on personal health care. In fiscal year 1975 this amounted to almost 31 billion dollars. If the proportion of the total remained the same in 1976, the dollar amount spent for personal health care of the aged was about 35 billion.

We need to know how much of this medical care benefits the people it was designed to help. Thus, evaluation of health services in terms of benefits to the elderly population is a prime area of concern.

There has been remarkably little hard conceptual work on the relationships between morbidity or frequency of disease and the utilization of health care. Without a conceptual framework, problems of definition and measurement are largely ignored; the data collected are frequently not those which are needed. Most discussions of health care implications focus on correlations between distributions of people and health care providers instead of on thorough knowledge of the determinants of the observed distributions in terms of cause and effect. For example, we say that since there will be more elderly people x number of years from now there will be more need for health resources. Is that necessarily true?

The lack of work on the determinants of health care utilization and on the relationship between utilization and morbidity is a particularly acute problem when the population at risk is the elderly because the levels of both morbidity and utilization are so high and because concern over the rising cost of health care suggests that there will be pressures for changes in legislation or regulations. Benefits under Medicaid are already being reduced in some States.

I would like to turn to the field of health administration for a model to use in first pointing out some complexities of the relationship between the need for and utilization of health care and then as a framework for listing areas where we have only fragmentary knowledge, no knowledge, or where we have not even defined what it is we need to know.

Donabedian created a model of the medical care process. It appears in *Aspects of Medical Care Administration* (3) and is one of several approaches which could be taken. This model (figure IV–3) shows several points beautifully. *First*, the relationship between need and utilization is not a simple straightforward one in which a sick individual seeks care, is treated, and is released as a well person; *second*, the entire process is embedded in the environments of both the individual and the provider (environments which frequently do not overlap) with their attendant sociocultural and organizational components; and *third*, health outcomes (modification of need) must be measured if the process is to be evaluated. The process is circular. Need influences utilization which in turn modifies need.

If we accept this model of the medical care process as a useful one, what can we do about filling in the boxes? What do we know about each of the components? What data do we have available about the aged?

The epidemiologist measuring the distribution of disease in the population knows to be alert to the difficulties and biases in the sources of data. The major sources of such data—death certificates, medical records, population based surveys, and administrative records developed for other purposes such as disability claims—all have limitations which are characteristic of that type of data (4). In addition, any specific data set has limitations peculiar to itself.

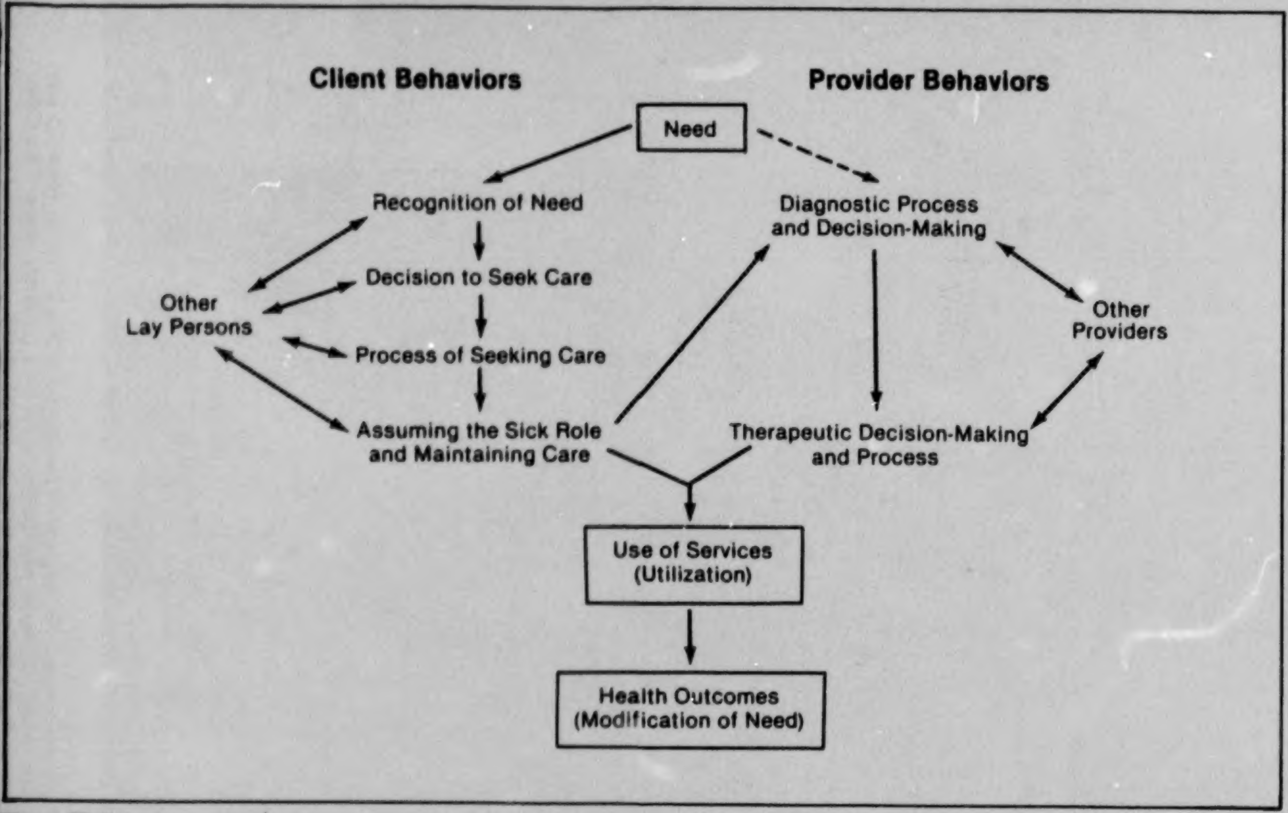
Client Behaviors
Provider Behaviors
Need
Recognition of Need
Decision to Seek Care
Process of Seeking Care
Assuming the Sick Role and Maintaining Care
Other Lay Persons
Diagnostic Process and Decision-Making
Therapeutic Decision-Making and Process
Other Providers
Use of Services (Utilization)
Health Outcomes (Modification of Need)

Figure IV–3.

A model of the medical care process

(Reference 3, reprinted with permission from Harvard University Press).

The individual attempting to measure need has to be aware of these limitations and of the critical distinction between the measurement of need and the measurement of disease, morbidity, functional disability, or other indicators of health status. Need for medical services exists if and only if the condition is amenable to medical intervention. (This is not meant to suggest that needs for other kinds of care do not exist.) Unfortunately, this distinction is not made frequently enough in population studies. Much of the time people conducting the studies are unaware of the distinction. Much of the research on measurement of need remains to be done.*

The distinction is important for any age group, but it is crucial for the elderly where the functional disabilities and impairments are results of conditions and events accumulated over many years. Medical care might have made a difference at the time an event occurred or when a condition was first active, but it may now be too late to restore the lost vision, hearing, or other function. In many cases, the present need may be for "caring" rather than for "curing." The curing functions of medical care should not be neglected, but the possibility of providing caring in other and perhaps better ways should be investigated. The existence of morbidity or disability, the need for care, and the need for medical care are not equivalent.

There is a great need for research on how the elderly person recognizes need for care, makes a decision to seek or not to seek medical care, and the process of seeking care. These are only special categories of behavioral and social science research, but they do deserve special attention because a delay in obtaining medical care when it is needed can result in suffering or death. Many elderly people may think "nothing can be done," they may be afraid that they will be told they are terminally ill or that they should no longer live alone. They may talk with friends or family or they may talk with no one. And if the process of seeking care from a fragmented medical care system is difficult for a relatively vigorous, relatively well educated young person what is it like for an older person who feels at a disadvantage socially and physically?

The final entry in the left-hand column pertains to the *sick role.* In a recent review of Parsons' sick role concept, Segall pointed out that there are many more questions than answers (11). How do behavioral expectations vary among different segments of the population? For which illnesses are people likely to consider themselves responsible? He reviewed the literature on applications of the sick role model to chronic illnesses, which are very obviously problems among older people, and stated that much work remains to be done.

In this context it is interesting to note that among people 65 or older, the proportions of men reporting poor health is only slightly higher than the proportion of women. However, the proportion of men "unable to carry on major activity" is over three times that for women.

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* Donabedian devotes 150 pages to "Assessment of Need." A few recent papers are listed in the references and papers have been published since. The Clearinghouse on Health Indexes lists work in progress (5–10).

Are the men accepting a sick role? Certainly hospital utilization rates which are higher for women than for men at younger ages are reversed.

On the other side of the diagram are the providers of medical services. It is intuitively obvious that the distribution and amount of health care resources, facilities and manpower influence utilization rates, but the nature of the relationship is only beginning to be explored.

The nature of the distribution of the providers and the reasons for that distribution are as yet poorly understood. In a free market, providers of services—shops, theaters, and other outlets—will appear when need or demand is great enough to support them and disappear when there is not enough demand. Demand is determined by the desire for the service or product and the ability to pay. The prospective purchaser can do some comparison shopping and make a decision based on quality and price.

For a number of reasons, the medical care market does not work that way. People sometimes choose to have a service—an annual physical exam or extra glasses—but very often they are sick and feel they have no choice; they must have medical care right now. It cannot be put off. What proportion of the medical care for the elderly goes into each category? Do not forget that even if the condition turns out not to be serious or is self-limiting without medical care, the patient does not know that without seeing the doctor.

Moreover, the ability to pay for certain services is not an overriding concern for the elderly population. In fiscal year 1975 when over $30 billion were spent for health care for people 65 and over, 90 percent of the hospital care and 59 percent of the physicians' services were paid for out of public funds. For other services—dental care, eyeglasses, and drugs—the major part of the expenditure comes from the individual so medical care for those services does depend more on ability to pay.

The distribution of medical care resources is influenced by factors other than need for services. A large teaching hospital, for example, tends to attract specialists thus draining them from other areas without much regard for the people's need for services. Areas which are culturally attractive also tend to attract doctors as do high income areas.

I do not know whether the aged are more affected by the maldistribution of physicians than are younger people. Barriers to medical care, that is inability to obtain medical care "when you needed it," are reported less frequently for the elderly than for younger adults. The elderly are more likely to have a usual place of care and less likely to utilize hospital emergency rooms. They are, however, more likely to rely on general practitioners who are evenly distributed throughout the population than are younger people. Whether the utilization of general practitioners is based on choice or availability, I do not know.

There is need for research on the diagnostic and on the treatment decisionmaking processes of the physicians. In many ways, older people are physiologically different from younger ones. Are those differences taken into account in making diagnoses? In prescribing the amount or kind of drugs? In deciding on how to treat or even whether to treat? Does the physician even have the knowledge to make informed decisions?

That question, it seems to me, is of critical importance. A high proportion of the visits to office-based physicians made by elderly people are to general practitioners and many general practitioners are themselves 65 or older. Dr. Butler has spoken of the need for training specialists in gerontology. Is there also a need for retraining the physician actually providing care so that currently available knowledge will benefit the people who need it now? Are the physicians making the decisions about how to care for older people operating on the premises they learned in medical school 30 or 40 years ago? I think that as you plan biomedical research in aging you need to recognize that many of the aged are receiving their medical care from that segment of the medical profession least likely to keep abreast of current knowledge. There is a need for research on the knowledge and attitudes of physicians in practice.

In the recent book, *The Effect of the Man-made Environment on Health and Behavior*, Hinkle states:

> One of the problems in all measurements of disability has to do with the ambiguity of social attitudes toward disturbances of mood, thought, and behavior. In general, there is agreement in this and other societies that episodes of disability caused by enviornmental agents, such as bacteria, viruses, physical force, and most chemical agents, . . . by the gradual failure of organ systems, and by genetic disorders are, in effect, "illnesses." But there is not complete agreement about when the disorders of the highest integrative function of the organism—those that are manifested by disturbances of mood, thought, and behavior—constitute "illnesses." When these disorders are profound, as in psychotic states, there is general agreement that they constitute "illnesses." On the other hand, when disturbed behavior is associated with sexual aggression, assault, or murder, this behavior may be regarded as a "crime." When disturbed behavior is evidenced by the stealing of property, or by the forging of checks, . . . this is regarded not as an illness, but as the result of voluntary behavior . . . (12).

When I read that passage, I wondered how often some of the disturbances of mood or thought of older people are regarded as either voluntary behavior or attributed to aging rather than as illnesses with causes and possible cures. How often are elderly people misdiagnosed as being senile and untreatable, rather than ill and treatable, because the most evident symptoms are disturbances of mood, thought, or behavior?

This raises a question about the need for mental health care and the utilization of services. I have been told, for example, that depression is very prevalent among the elderly. I do not know how to evaluate that statement but I am prepared to accept it. There are drugs and illnesses which have a depressing effect. The loss of a spouse and lifelong friends, loss of income, moves to other housing, or retirement are all real and may trigger a depressive reaction. Yet utilization of outpatient psychiatric facilities is lower among the aged than among other adults. Are physicians failing to recognize that depression along with many other emotionally disabling conditions can be treated?

The decision made by the physicians have more influence on the utilization of services once the physician has accepted the patient than decisions made by the patient. The patient can terminate treatment but has little voice in the kind of treatment or whether it is provided on an ambulatory or inpatient basis. In many cases, how many I do not know, the physician bases his decision at least partly on his knowledge of what Medicare and Medicaid will pay for. The care may not be the most appropriate but, out of consideration for the patient's financial state, the short-stay hospital followed by a nursing home may be the choice instead of office visits and home care. Surgery and drugs are paid for in the hospital. Rehabilitative therapy and drugs outside the hospital may not be. How much of the medical care provided for the elderly is appropriate? How much of the medical care is utilized when, if payment mechanisms were available for all forms of care, other means of providing help would be better for the aged person and would take some of the burden off the medical care system?

Finally, the care provided may influence the outcome. In some cases the patient will be cured. In some cases symptoms will be relieved or a condition kept under control even though cure is not possible. In other cases there will be no change in the need for care either because the patient did not respond or because the care provided was not appropriate. Vast quantities of time and money may have been spent because the physician did not know how best to treat an elderly patient or very little may have been spent because he did not try. Far worse, however, is the possibility that the patient may be worse off. He may have increased suffering and pain; he may be dead. The person given a drug which was clinically tested in a young adult population but which has an adverse effect on an elderly person has not benefited from medical care nor has the patient who functioned reasonably well in the community but whose placement in a nursing home resulted in depression and dependency.

We need to know far more than we do about levels of morbidity and functional disability among aged people. We need to know the etiology so that we can distinguish between the visual impairment that can be treated by cataract surgery and the one that cannot, the limitation of mobility which can be alleviated by arthroplasty and the limitation due to untreatable paralysis. One indicates a need for medical care while the other suggests the need for other kinds of help.

We need to know how the aged make their decisions to seek care or not to seek care. (Twenty-one percent of the elderly people have not seen a physican with a year according to the Health Interview Survey. Sixteen percent of the people continuously enrolled in Medicare from

July 1966 to December 1974 never met the supplementary medical insurance (SMI) deductible, according to Medicare records.) Having made a decision to seek care, how do they decide on a physician and adapt themselves to taking advice and following treatment?

Does the distribution of medical manpower influence these decisions of the individual? Does it influence the amount and quality of care provided? How does the physician make his decisions? How much is he influenced by lack of knowledge, lack of time, lack of professional contacts, and his own biases?

Does the care provided really benefit the patients? Are they in better condition physically and emotionally because they have utilized the medical care system?

Is medical care really the most appropriate form of care for many of the problems of the elderly? Are we making unrealistic demands on the medical care system?

Those are only some of the questions. Perhaps they will suggest ideas for research which is directed toward maintaining not just a large population of elderly people but a large population of healthy functioning elderly people.

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Discussion

Gruenberg: We have discussed at length the mechanisms by which medical care can be an epidemiological force affecting the frequency of uncorrected conditions, creating the iatrogenic and nosocomial conditions. I wish we would add only one thing and that is some estimate of what the doctor would have if he were fully confident and using good judgment in each case. What is the nature of the tool box? What is the technique available? Which conditions can be corrected? Which conditions cannot be corrected today?

Kovar: I don't have good answers to those questions. The point I hoped to make was that doctors operate within the limits of their own capabilities and environments. Many doctors treating older people may not be practicing at an optimal level because they do not have the benefit of recent medical knowledge, because they are rushed, or because they don't practice in a medically sophisticated environment. It is not enough that major medical centers be repositories of knowledge. I want the doctors who are actually treating elderly people to know how best to care for them. And the evidence is that many doctors now practicing have not been exposed to modern geriatrics. I think that extending knowledge to the practicing physicians who provide the care to the elderly will do more toward improving the health of the elderly than a technological advance.

Gruenberg: As outcomes, you do not include perpetuating disease by curing fatal complications which is perhaps the most powerful thing that medical care does with the distribution of disorders today. It thwarts the fatal complications and thereby perpetuates chronic illness and raises the prevalence of disease and affects morbidity by that mechanism.

Kovar: It can certainly raise the level of the prevalence of disease. That does not mean that the level of disability associated with that disease is necessarily raised. There are a number of levels of measurement in morbidity. We have usually not spelled them out concretely because we have not thought enough about what it is we want to know. For different purposes we may want to know the prevalence of a chronic condition by medically accepted criteria, the prevalence of a chronic disability by criteria which establish level of disability, the prevalence which current medical knowledge could alleviate, and the prevalence currently receiving medical care.

Siegel: You seem to be saying that what we need in morbidity research is a knowledge, attitude, and practice (KAP) study like those we have carried out in fertility research.

Kovar: I did not say we need KAP studies, however, I thought of them as I wrote the paper.

Siegel: We need a KAP study addressed to the patient or the general population.

Kovar: I was primarily addressing myself to the physicians or other people providing care. We have heard many stories about the attitude of medical providers, but we really have only fragmentary knowledge. What would a KAP survey reveal about the knowledge, attitudes, and practices of the medical professionals who are *actually* providing primary care?

Wilkie: Years ago I conducted a national study which was concerned with the reasons why people did or did not give medical care to older people. At that time, we were making every effort with all kinds of questions to find out the number of people who were being denied medical care or denying themselves medical care because of financial reasons. The two major reasons which came out then were "I do not like to bother the doctor," and "What can the doctor do for me?" Now, that was 20 years ago and I have not repeated that particular study because other than you and the people at the National Center for Health Statistics, I have not heard anyone interested in this topic. They assume that all the problems of the aged should be medical care and now they have Medicare. But I would guess that if we asked older people about their complaints and their physician visits, the same reasons would be given.

There is only one factor that concerns me a little about that. Although this is my overall guess, I think we are getting a more medically sophisticated group in the older age group. That is to say, the people who are 65 to 69 have had some exposure to preventive medicine, i.e., early examinations, and that may make a difference.

We may be getting a group of people coming into this older age group who take seriously the admonition to "see your doctor once a year." If that is indeed true, it may make a difference. If that is not true, I still think you would get the same thing from older people: "What can he do for me? Besides, I do not want to bother him."

Kovar: I am also concerned about the doctor who thinks there is nothing he can do.

Wilkie: The National Center for Health Statistics had one category in a medical record report called "old age senility." If you value the view of the center, which is ostensibly quite sophisticated in this area, what can you expect from some beknighted physician?

Kovar: I can hope that the "beknighted physician," when admitting a patient to a nursing home, will record a more specific diagnosis than old age or senility. The NCHS can only publish what is reported to it. The table you refer to is based on data collected from nursing homes and their records, not on our own physical examination of the patients.

Feinleib: You might be interested in the question of attitudes and practices of medical personnel. I recently did a survey of the literature and found 12 different studies which consistently showed that medical students, doctors, nurses, psychiatric personnel, and social workers generally accepted most of the negative stereotypes about the aged and preferred not to treat the aged and so forth.

Kovar: If I understand, you are saying that students as well as professionals in practice have negative attitudes.

Atchley: I think that the Harris poll and a number of other surveys have shown that the most consistently negative attitudes that older people have is toward medical care institutions of all kinds including physicians, nursing homes and hospitals. They are very skeptical that anything can be done for them.

Shanas: Then there have also been studies of attitudes of medical personnel toward the dying which are very negative.

Huycke: There was a conference at the National Institutes of Health on geriatric medicine and I think the whole point of that conference addressed this question—how to educate doctors and health personnel to deal differently with the older person. Hopefully, we will see a change.

Organization and Financing of Health Care for the Aged: Future Implications*

Henry P. Brehm, Ph. D.

Office of Research and Statistics
Social Security Administration
Meadows East
6401 Security Blvd.
Baltimore, MD 21235

In attempting to discuss the changes likely to occur over the next 25 years or more in the delivery and use of health-related services for the elderly, certain assumptions have to be accepted. First, we start with recognition of a continuing national commitment to the concept of the right to health and health care unimpeded by personal financial resources. Second, although we cannot anticipate what scientific or medical breakthroughs may occur at the level of the content of care, we can discuss the potential future impact of expected demographic changes on the delivery and use of health-related services for the elderly, of certain proposed changes in the organization and financing of health care, and of extrapolating trend lines in utilization patterns and other related social and economic factors.

Impact of Demographic Changes on Health Care Delivery

The demographic changes we can expect in the next 25 years or more have been discussed in detail at this conference by Jacob S. Siegel of the Bureau of the Census. Changes in the age distribution of the U.S. population which might have an impact upon the health care system and the delivery of health care for the elderly will be summarized here. Reference will be made to Tables IV-1 through IV-3 of the Conference Proceedings.

Using 1976 as a reference year, the U.S. had a more dramatic rate of increase in its aged population from 1950 to 1976 than is projected for the period betwen 1976 and 2000. As a percentage of the population, the aged (65 years and over) comprised 8.1 percent in 1950; this rose to 10.7 percent in 1976 and is projected between 11.3 percent and 12.9 percent in the year 2000, depending on which set of fertility assumptions is used (Table IV-2). Using the Series II projections as the probable best guess provides an estimate of 12.2 percent aged 65 and over. The data indicate there has been more of a change in the proportion of the aged population in the 1950 to 1976 period than is projected over the next 25 years. The rate of change in the size of the population 65 and

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over was also more dramatic from 1950 to 1976, where it showed an 85 percent increase (12,397,000 to 22,934,000), than from 1976 to 2000 where it is projected, under all assumptions, to increase by 39 percent (22,934,000 to 31,822,000) (Table IV–1).

In evaluating these data, it must be remembered that as subsequent base years become larger, a constant increase in numerical size will show a decreased rate of increase. However, when we look at the increase in absolute number of aged persons, we see a larger increase from 1950 to 1976 than from 1976 to 2000. The last 25 years has seen a growth in the number of aged persons of approximately two million every five years. The next 25 years will see approximately the same level of increase until 1990, when it will drop and remain at a lower level of increase until after the year 2005. At that time, it will substantially increase again due to different size birth cohorts from 1940 on.

Even with these increases in the numbers of aged persons, their proportion in the population is projected to be only nominally larger by the year 2000 because of the anticipated growth in the overall population. Thus, while the numbers of aged persons will grow by 39 percent from 1976 to 2000, the overall population will grow by 23 percent. From 1950 to 1976, the aged portion of the population grew at twice the rate of the overall population (85 percent and 40 percent, respectively). Between 2000 and 2020, the rate of increase in the proportion of persons 65 and over will be similar to that between 1950 and 1976. By the year 2020, 15.5 percent of the population will be aged 65 and over.

The age distribution within the 65 and over age group is expected to shift between now and the year 2000. The population 75 and over will grow at a faster rate than the population 65 to 74. In 1976, 4.1 percent of the total U.S. population was 75 and over. In 2000 it will be 5.5 percent (Table IV–2). Within the population aged 65 and over, 38.2 percent was 75 and over in 1976; by 2000 this will grow to 45.2 percent (Table IV–3). The absolute numbers aged 75–84 will grow by 56.9 percent by the year 2000 computed against 1976 as a base (Table IV–1). For the 85 and over age group, the comparable figure is 91.0 percent. These figures can be compared to the projected increase for the entire group 65 and over which is, as indicated above, 39 percent for 1975 to 2000.

These data do point to a definite increase in the number and proportion of elderly persons in the U.S. population in the next 25–45 years. However, the data show the rate of change will be less abrupt in the next 25 years than it has been in the past 25 years. The U.S. population will be expected to take on more elderly persons between now and the year 2000, but at a basically lower rate of increase, both in absolute numbers and as a proportion of the total population, than has been true since 1950. It is the population aged 75 and over that is projected to show the most dramatic increase in number relative to its present size. However, this will result in an increase proportion of just over one percent of the total population (4.1 to 5.5 percent) in a period of 25 years. The increase is only sizable relative to the present absolute number of persons in that age group.

The question which concerns us is whether this increase in the population age 65 and over, or in subparts of that population, can be expected to require or produce any changes in health-related services in the near or middle range future? Such a question cannot be considered solely from the perspective of the projected population changes. Unlike some other areas of social program and service delivery, health services are not an exclusive domain of need or use among the aged or even one primarily oriented to meeting the demands of the aged. All age segments of the population make demands on the health care delivery resources of this country, albeit in differing amounts and with differing requirements. On the other hand, public income maintenance programs traditionally are oriented primarily to support of the aged and dependent children, with special exceptions made for those in between who are considered unable to work. Changes in the ratio between working age persons and the aged and the overall dependency ratio are critical for assessing the potential burden of such support to be borne by each working age person. However, the provision and use of medical care services is a somewhat different situation. In this area, the issue is the differential rates of use and patterns of use by different age groups and the impact that changes in the age distribution and the relative number of persons in certain age groups will have an overall utilization and utilization of specific services and facilities.

If programs such as Medicare and Medicaid were the only ones at issue, then dependency ratios would be of concern for the public financing of medical care for the aged. However, we anticipate the passage of some form of national health insurance before the end of the century. Even the current debates have centered more on specifics than on the principle of whether responsibility for the financing of health care should be borne by the population at large through government involvement. Under these circumstances, resources allocated for the public financing of health care use by the aged as compared to other age groups is proportional to the relative use and cost of health care by different age groups. The working age population will bear the major burden for the financing of all such care paid for with public funds. Costs for the health care of the elderly will increase this burden to the extent that their costs become a higher percentage of the cost of all health care and/or there is an increased percentage of the gross national product devoted to health care.

The trends in the use and cost of health services by the aged will be reviewed relative to those in other age groups. Changes in patterns of use over time are shown in table IV–13, A–F.

Physician Visits.—Table IV–13–A shows that physician visits per person per year were 5.0 in 1973, for all age groups. For those age 65 and over, the rate was 6.5. The differences between age groups in physician visits for those persons over age 45, if anything, have become smaller since 1963. For the total population there has been an increase

Table IV–13.

Summary data on use of health services: comparisons among age groups and over time

A. Physician visits per person per year

	July 1963 to June 1964	July 1966 to June 1967	1969	1971	1973
All Persons	4.5	4.3	4.3	4.9	5.0
Age:					
Under 5 years	5.5	5.7	5.7	6.8	6.5
5 to 14	2.8	2.7	2.8	3.3	3.4
15 to 24	4.3	4.0	3.7	4.5	4.5
25 to 34	4.7	4.4	4.4	5.1	5.3
35 to 44	4.4	4.3	4.1	4.5	4.9
45 to 54	4.8	4.3	4.3	5.1	5.4
55 to 64	5.3	5.1	5.1	5.9	5.5
65 to 74	6.3	6.0	6.1	6.4	6.5
75 and over	7.3	6.0	6.2	7.2	6.6

B. Admissions per 1,000 total population: general and special hospitals

	1950	1955	1960	1965	1966	1967
Admission rate	110	125	136	146	146	148
Average length of stay: in days	10.6	9.9	9.3	9.1	9.5	9.1

C. Admissions per 1,000 person-years: 1963

		Ages					
	All ages	0 to 5	6 to 17	18 to 34	35 to 54	55 to 65	35 and over
Admission rate	130	80	60	190	140	170	180

D. Short-term general hospital (non-Federal) utilization, persons aged 65 and over

	1967	1968	1969	1970	1971
Admission rate per 1,000 aged persons	275	285	301	310	314
Average length of stay (days)	12. 7	13. 3	13. 2	12. 8	12. 4

E. All hospital inpatient admissions, hospital insurance enrollees under Medicare

	1969	1970	1971	1972	1973
Admission rate per 1,000 HI enrollees	307	307	305	313	320
Average length of covered stay (days)	—	13. 1	12. 6	12. 1	11. 7

F. Persons hospitalized per 1,000 population, by race and age, selected years

	All ages			Under age 15			Ages 15 to 44			Ages 45 to 64			Ages 65 and over		
	1961–62	1966	1968	1961–62	1966	1968	1961–62	1966	1968	1961–62	1966	1968	1961–62	1966	1968
White	95	103	97	52	58	46	125	125	113	98	112	102	114	134	158
Black and other races	73	81	83	36	43	39	114	120	116	68	83	90	78	88	126

Source:

A. Table B, "Physician Visits," Vital and Health Statistics, data from the National Health Survey, National Center for Health Statistics, series 10, number 97, March 1975.
B. Table 161, "Pocket Data Book, USA 1969," U.S. Department of Commerce, Bureau of the Census.
C. Ronald Andersen and Odin W. Anderson, "A Decade of Health Services; Social Survey Trends in Use and Expenditures," Chicago: University of Chicago Press 1967.
D. Table 8, "Five Years of Medicare—A Statistical Review" by Howard West, "Social Security Bulletin," vol. 34, no. 12, December 1971, pp. 17–27.
E. Medicare, Fiscal Years 1969–1973, Selected State Data, Social Security Administration, DHEW Publication No. (SSA) 76-11711.
F. Table 7, See article by Karen Davis, "Milbank Memorial Fund Quarterly," fall 1975.

in average visits per year since 1966–67, after a small drop from 1963–64 to 1966–67. For those 65 to 74, the pattern is more stable and shows a smaller increase. For those 75 and over, there is an irregular pattern which does not show a net increase over the decade.

Since use by those under 65 has been increasing more than by those 65 and over, it is unlikely that the projected increase in the proportion of the population which is over 65 will mean that older people's share of physician use will increase in the next 25 to 45 years.

Hospital Care.—There are more pronounced differences in hospital services usage both among age groups and over time. The rate of admission to general and special hospitals per 1,000 persons in the total population increased from 110 in 1950 to 146 in 1965, the last full year before the Medicare amendments to the Social Security Act went into effect, and to 143 per 1,000 in 1967 (table IV–13–B).

In 1963, well before the passage of Medicare, there was a rate of 180 admissions to short-term hospitals per 1,000 person-years for those age 65 and over compared to a rate of 130 per 1,000 person-years for the total population. With the exception of the increased hospital admission rate for persons in the child-bearing years, there was a definite trend of increased hospital use with age (table IV–13–C).

For the population 65 and over, the rate of admission to short-term general hospitals was 275 per 1,000 population in 1967 which rose to 314 per 1,000 in 1971 (table IV–13–D). For the Medicare enrolled population, the trends are quite consistent—in 1969 the hospital admission rate was 307 per 1,000 enrollees which rose to 320 per 1,000 in 1973 (table IV–13–E). Two trends are evident in these data. There is a higher rate of hospitalization for those age 65 and over than for younger members of society; and the hospital admission rate for those 65 and over has grown substantially over the years, particularly from the period before to after Medicare. At the same time hospital admission rates have been increasing, average lengths of stay have been generally decreasing after an up-swing for the aged in average length of stay for the first few years after Medicare went into effect (table IV–13–D and E).

In an analysis of the distribution of Medicare benefits by the socio-economic characteristics of elderly persons, Davis (1) points out that after the advent of Medicare there was a decrease in the disparity in the rate of hospitalization of blacks as compared to whites. The implication is that at least as regards this one area of medical care, the reduction of a financial barrier to care altered the pattern of use for a group which has traditionally had lower levels of utilization.

According to table IV–13–F, it is apparent not only that there was a differential rate of change in the use of hospitalization before and after Medicare between the races, but that there was a pronounced difference in the rate of persons hospitalized per 1,000 population (as distinct for admission rate) by age groups. For those under age 65, the pre-to-post-Medicare rate of persons hospitalized shows a decrease after

Medicare. For those 65 and over, regardless of race, there is a substantial increase.

These presumed effects of Medicare point out the degree to which patterns and trends in medical care usage may be affected by a change in the financing system for medical care.

Nursing and Personal Care Homes.—Nursing and personal care homes are a special category of health-related facility used in overwhelming measure by the elderly. In 1963, there were 505,242 residents in nursing and personal care homes in the United States. Of these, 11.8 percent were under age 65 (see table IV–14) and 70.4 percent were aged 75 and over. The number of residents per 1,000 population was 0.6 for the population under age 65, 57.1 for those age 75 and over, and 148.4 for those age 85 and over. By 1969, after the Medicare and Medicaid programs were well into operation, the age distribution of residents in nursing homes was not much different from what it had been in 1963,

Table IV–14.

Summary data on residents in nursing and personal care homes[1]

	1963		1969	
	Number	Percent	Number	Percent
Number and percent distribution of residents in nursing and personal care homes, by age				
Total	505, 242	100. 0	815, 100	100. 0
Age:				
Under 65	59, 678	11. 8	92, 900	11. 4
65 to 74	89, 619	17. 7	138, 500	17. 0
75 to 84	207, 243	41. 0	321, 800	39. 5
85 and over	148, 702	29. 4	261, 900	32. 1
Number of residents in nursing and personal care homes per 1,000 population by age				
All ages—20+[2]	4. 5		4. 0[2]	
20 to 64[2]	0. 6		0. 5[2]	
65 to 74	7. 9		11. 1	
75 and over	57. 1		7 . 6	
75 to 84	39. 6		([3])	
85 and over	148. 4		([3])	

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[1] For 1963 data, table B and table 2 "Characteristics of Residents in Institutions for the aged and chronically Ill, U.S.—April-June 1963," Vital and Health Statistics, data from the National Health Survey, National Center for Health Statistics, series 12, No. 2, Sept., 1965. For 1969, table D and table 1 "Characteristics of Residents in Nursing and Personal Care Homes, U.S.—June-August 1969," Vital and Health Statistics, data from the National Health Survey, National Center for Health Statistics, series 12, No. 19, Feb. 1973.

[2] For 1969, these rates are based on the entire population of concern and do not exclude those under age 20.

[3] Data not available for 1969.

but the residents per 1,000 population was dramatically higher, particularly among the very old. It was 0.5 per 1,000 population for those under 65, but had increased to 76.6 per 1,000 population for those 75 and over. The residents per 1,000 population had gone up for those age 65 to 74 from 7.9 in 1963 to 11.1 in 1969. There was an overall increase in the number of residents in nursing and personal care homes from 1963 to 1969—309,858 to 815,100, respectively.

In absolute numbers, the population of these homes had grown by 227,755 persons age 75 and over between 1963 and 1969. If we apply the 1963 rates of residents per 1,000 population to the 1969 population for the 75 to 84 and 85 and over groups, the growth in the nursing home population would have been only 81,932. The remainder of this increase is based on the change in pattern of use and not the increase in the size of the very old population. If we apply the 1969 rates to the projected year 2000 population aged 65 and over, by age group, we will need space for an additional 503,086 elderly persons in nursing and personal care homes. This would be a much slower annual rate of increase in residents from 1969 through the year 2000 than happened from 1963 to 1969. Between the years 2000 and 2020 the annual rate of increase in residents of these homes would still be projected to be slower than it was between 1963 and 1969, even assuming a sizable increase in the proportion of those 65 and over who are 75 and over. Accommodating such increases should be no less manageable than the change from 1963 to 1969, whether by increased capacity use or the building of new capacity. If the rate of residents per 1,000 population increases as dramatically in the next 25 to 45 years as it did from 1963 to 1969, this will create an entirely different situation. However, the 1963 to 1969 increase in number of very old people in nursing and personal care homes was not primarily the result of more elderly people in our society, but of an increase in their rate of use of these homes. Such a pattern and its trend of change over time is a medical care delivery system factor, not the result of population changes, and is, therefore, potentially controllable if there is a desire to exercise control over the delivery system, its use, and the total of economic resources devoted to it.

Cost of Health Care.—These patterns and trends in the use of physicians, hospitals, and nursing and personal care homes, in combination with inflation rates, have significantly affected the overall and the per capita levels of expenditures for health care and the percent of the gross national product used for health-related goods and services over the last several years. Table IV–15 shows the national health expenditures and the changes that have taken place since 1950. The total national health expenditure in fiscal year 1950 was $12,028 million which amounted to 4.6 percent of the gross national product. Preliminary estimates for fiscal 1974 show a total expenditure of $104,239 million or 7.7 percent of the gross national product. The per capita expenditure rose in the same time period from $78.35 to $485.36. If we apply the 1950 per capita figure to the 1974 population, there would have been an increased total expenditure not of $92 billion, but of $4.8 billion. In other words, slightly over 5 percent of the total increase in expenditure can be attributed to population growth, the rest is the result of a combination of inflation in unit costs and increases in levels of utilization.

The major specific items of increase in per capita health expenditure from 1950 to 1974 were for hospital care, which went from $24.09 to $190. 44, and nursing home care, which went from $1.16 to $34.69. When we look at the change in per capita costs for personal health care for the elderly from just prior to Medicare to several years after Medicare, we see a pattern of continuous increase year-by-year of approximately $100 a year in per capita from $445 in fiscal 1966 to $1, 218 in fiscal 1974. Again, hospital and nursing home care showed the steepest increases, from $177.84 and $68. 39, respectively, in 1966 to $573.18 and $289.10 in 1974.

Impact of Medicare on Health Care for the Elderly

The data on use and cost of health services indicate that the increases in demand and expenditures for such services over the past 10 to 25 years have primarily been the result of changes in the pattern of use of these services and dramatic increases in their cost. This occurred in a time period when the increases in the absolute numbers and relative proportions of the aged in our population took place at a more dramatic rate than is projected over the next 25 years. Unquestionably, some, but by no means all, of these increases were related to the advent of Medicare and Medicaid. The history of increases in the levels of use, and percent of the gross national product and per capita expenditures for health care attest to this fact. Given this record of changes in the use and cost of health related services over the last several years, it is probable that our ability to rationalize the use of services and to exercise some control over the organization for their delivery and financing will be more significant than changes in the elderly population itself.

Aside from these indications of the impact of Medicare on the use and cost of care for the aged, there are other data available as to its effect on the system for delivery of care. In a study of the health delivery system in Kansas City (*2*) comparing the situations just before and 2 and 4 years after Medicare, no appreciable change was noted in the organization of health care services. Most physicians had not altered their methods of practice although they recognized the need for other levels of care. Few plans to expand facilities and services made just after Medicare was implemented were actually carried out and no further Medicare-related change in services was contemplated. There was no coordination between nursing homes and hospitals.

These findings ran counter to the implicit, if not the explicit, intent of Medicare which was to affect the provision, organization, and patterns of referral for health services for the aged within a community's health care subsystem. It was expected that Medicare would result in an expansion of facilities and services and their coordination to provide more comprehensive services for the elderly including transitional forms

Table IV–15.

Summary data on national health expenditures

Aggregate and per capita national health expenditures and percent of gross national product, for fiscal years 1950 and 1974

Fiscal year	Amount (in millions)	Per capita	Percent of GNP
1950	$12, 028	$78. 35	4. 6
1974	104, 239	485. 36	7. 7

Per capita expenditures for selected national health services for fiscal years 1950 and 1974

Fiscal year	Hospital care	Physician's services	Nursing home care
1950	$24. 09	$17. 52	$1. 16
1974	190. 44	88. 47	34. 69

Estimated per capita personal health care expenditures in total and for selected items for population 65 and over fiscal years 1966–1974

	1966	1967	1968	1969	1970	1971	1972	1973	1974
Hospital care	$177. 84	$223. 58	$282. 89	$335. 76	$375. 13	$418. 55	$468. 61	$508. 93	$573. 18
Physician's services	89. 57	108. 97	122. 40	127. 49	141. 60	154. 37	172. 58	180. 74	182. 14
Nursing home care	68. 39	84. 94	113. 56	133. 18	162. 76	202. 39	237. 79	265. 11	289. 10
Total	445. 25	535. 03	646. 65	735. 19	828. 31	925. 98	1, 033. 51	1, 119. 78	1, 217. 84

Source: "*Compendium of National Health Expenditures Data,*" U.S. Department of Health, Education, and Welfare, Social Security Administration, Office of Research and Statistics DHEW Pub. No. (SSA)76-11927, January 1976, Compiled by B. S. Cooper, N.L. Worthington and M. F. McGee.

of inpatient care (extended care facilities) and ambulatory care services. The referral pattern among providers was also expected to change and expand to enable continuity among various types and levels of care.

In the study by Davis (*3*), she concludes that "a uniform medical care financing plan has not been sufficient to guarantee equal access to medical care for all elderly persons. Those elderly population groups with the poorest health are the lowest utilizers of medical care services under the program—the poor, blacks, and residents of the South. Furthermore, differences on the basis of income, race and location are of sizable magnitude."

Davis also notes that, "the structure of the Medicare program, through its reliance on uniform cost-sharing provisions for all elderly persons, is largely responsible for the greater use of medical services by higher income persons." She recommends that, "several changes in the Medicare program are required to reduce the inequities revealed by the current distribution of benefits. Four areas which seem particularly in need of reexamination are (1) The cost-sharing structure of Medicare, (2) efforts to improve access of minorities to medical care, (3) the sources of financing for Medicare, and (4) the method of physician reimbursement."

Suggestions for the general direction in which we should be heading were made by Coe and Brehm who noted:

> Since a considerable amount of research has shown time and again that our present fee-for-service organization of medicine is dysfunctional in terms of providing certain groups with adequate medical care—especially the poor and the aged—it would seem reasonable that some form of subsidized care would be provided. Medicare and Medicaid, of course, already provide some precedent for this approach. But these programs are not oriented to altering the present system for delivery of medical care. They provide financial support for the present system of delivery which is oriented primarily toward dealing with illness, not promoting health. What is needed is a combination of subsidized care and a reorientation of the delivery system to a coordinated comprehensive system concerned with the maintenance of a maximum health condition and preventing, as well as dealing with illness. Such a reorientation coupled with a reorganization of the delivery system would incorporate the physician into an organizational network which coordinates his efforts with those of other medical and paramedical specialists. This would relieve the doctor of the need to spend his time performing tests and other functions which can be handled by specialists and technicians with lesser levels of training. It could also alter the existing concepts of needed physician/patient ratios thereby easing some of the upward pressure on the

cost of medical care and partially relieving the shortage of fully trained physicians (4).

Alternatives to the Organization and Financing of Health Care Delivery

Among the systems being considered and actively pursued are some form of National Health Insurance, wider availability of Health Maintenance Organizations (HMO's), and increased use of physician extenders, nurse practitioners, etc.

National Health Insurance.—It seems reasonable to expect that the United States will ultimately adopt a national health insurance measure for the financing of care. However, it is also highly probable that our first such program will strictly provide a mechanism to finance the use of health care on a cost-sharing basis, with part of the cost of care paid by the user and part by the government/insurer.

HMO's.—The need to provide more coordinated, comprehensive health care services and at the same time contain the rapid increase noted in health care costs has given rise to a series of planning and experimental techniques. The HMO Act of 1973 and the inclusion of HMO's under the Medicare reimbursement program are two mechanisms to encourage this prepaid group practice arrangement as a potentially more effective and cost-conscious delivery approach.

While these mechanisms are designed to promote HMO's, there is some concern that various of the requirements placed on them to qualify for Federal funds may be restrictive of their competitive position. Efforts to promote HMO's are based on data which recognize their potential to alter patterns of use, particularly by replacing inpatient services with ambulatory care services and thereby reducing the overall costs of care.

As Harris points out in his study on the effect of bed supply on hospital utilization:

> The results of this study support the contention that hospital beds tend to create their own demand for utilization, at least for the New York State counties studied. While this elaboration analysis was based only on New York State data, additional evidence from other areas are consistent with its results. It is generally recognized, for example, that pre-paid health insurance plans, such as those of Kaiser-Permanente in Oregon and California, maintain fewer beds per 1,000 of their subscribers than is the case for more conventionally insured populations, and that these plans have lower hospital utilization rates than more conventional plans (5).

Physician Extenders.—Experimental programs are being initiated to promote the use of physician extenders. The same amendments to the

Social Security Act which provide the mechanism for incentive and prospective reimbursement experiments under Medicare as a way of investigating the potential for encouraging cost control in hospitals also provide the mechanism for programs using physician extenders. The intent is to promote the use of personnel who can substitute for direct physician services thereby increasing the availability of physicians for more complicated tasks by using lower paid personnel.

All of these various possible approaches relate to the need to deal with the overlapping problem areas of manpower provision, cost-financing, and availability of facilities and services at appropriate levels of care. Sound planning is obviously required to provide the coordination needed among providers to deal effectively with these problem areas. Klarman (*6*) points out that, "The primary reason for health planning in this country is the numerous instances in which the interests of the individual, health-care institution and those of the community may diverge. . . From a technical standpoint, it is much more difficult to plan for health services at the local level than nationally. Notwithstanding, health services are mostly provided at the local level. . . In the past decade, local health planning has been hampered by unstable federal funding. The absence of national policies and guidelines has lead to a constant quest for new ideas. . . What is required, in addition to steadier funding, is a fostering of local capabilities for health planning. . . In specified circumstances, the Federal Government is expected to serve as the superseding decisionmaker.

Future Outlook and Research

In view of the data presented on projected demographic changes, the patterns of use of health-related services and facilities, and the trends in the use of these services and facilities, what changes can be anticipated for the delivery of health care services for the elderly and what are some of the related research questions? Within the next decade it is expected that the U.S. Government will have adopted a National Health Insurance program. The program will carry provisions for deductibles and coinsurance and specified service exclusions. In addition, no greater exercise of control over the system for delivery of health care services can be expected. However, there will be an improved standard of health insurance coverage for the entire U.S. population. With an improved guarantee of payment, the general population will probably place a greater demand on the health resources of this country, with possible resultant downward pressure on the availability of health services for use by the aged. Alterations in overall demand based on this insurance program will probably not seriously erode the availability and use of health services by the elderly, but to the extent there is an effect, it can be anticipated to be a negative one for the aged population. Existing coverage under private health insurance policies and public medical assistance programs will lessen the impact that National Health Insurance might have had on levels of demand for health services from the population at large. However, the implementation of such a program should be monitored for its impact on the availability and use of health services, particularly those most appropriate for the type of health care needed by the aged.

It can also be expected that HMO's will become increasingly popular and serve as a mechanism for providing coordinated, comprehensive care not only for the aged but for all age groups. Fully packaged prepaid group plans which own and operate their own hospitals, skilled nursing facilities, etc., will be more common. Research will be needed to establish the value of these plans to contain costs and hold down utilization, particularly of the more expensive inpatient facilities without reducing the quality of care. The impact on cost and use of different organizational patterns and physician reimbursement mechanisms both within and outside of such HMO's should be evaluated.

Physician extenders, physicians' assistants, and associates, nurse practitioners and whatever new titles may surface to designate these categories of alter egos and limited replacements for physicians can be expected to come into greater use. They will serve as a means of spreading the supply of physician services over a larger population base by the use of lowerpaid, less highly trained personnel to deal with situations not requiring the physicians' level of knowledge and skill.

Additional research must be undertaken as to the most effective means to utilize these personnel, so as to increase the overall quality of care while reducing the demand for physician services and the costs of care. Additional problems still remain in institutionalizing the use of such personnel within the delivery system. More research is needed on such problems as mechanisms for financing and payment; the requirements for licensure and the permissible functions for such persons as they exist and differ from state to state; malpractice insurance complications such as whether the physician supervisor or the extender bears responsibility; and general acceptance by both physicians and patients of such staff and the nature of their functions so as to promote the smoothest transition to maximum use. Concerns for cost control and appropriate use of the hospital should promote wider use of a variety of care levels, including skilled nursing facilities, out-patient ambulatory and day bed care services, and home health care as a means to shorten or avoid hospital stays.

Research is needed on the efficiency and effectiveness of alternate treatment modalities and levels to control costs of care. Analysis must be undertaken of the organizational factors, financing mechanisms, and decisionmaking control elements as they operate within and between provider units to influence general measures such as rates of use, average length of stay, unit-patient cost, cost of an episode of care, and total population or area health care costs. Patterns of use of alternate treatment modalities and levels as these relate to the total costs of care, and the impact of changes and improvements in medical care technology on costs and efficiency in health care delivery must also be assessed.

These various organizational and financial mechanisms are already in use or about to be used. Their use and acceptance should expand beyond the very limited scope that exists at present for some of them. The next decade will probably not be one in which new ideas for the organization and delivery of medical care in general or for the aged are expounded. It will probably be a period of heightened awareness of the need to more effectively utilize medical resources and manpower and to control the cost of medical care by putting into practice the various delivery system organization and financing ideas which have been discussed over the last few decades. There is no shortage of ideas in this area. However, there are limitations in authority to direct the implementation of various delivery approaches and to coordinate or control the health care delivery system, and a deficient base of knowledge as to which mechanisms and organizational combinations will most effectively accomplish these purposes. The research questions reviewed must be addressed so as to expand this base of knowledge.

These anticipated changes in the general structure of the health care delivery system over the next few decades is one of movement along already defined lines. Health care for the aged will be affected more by these general developments than by changes in the age structure of the population. The adaptations needed in the health care delivery system to adjust to projected demographic changes in population should not require any drastic or concerted efforts. The projected population changes over the next 25 years will take place gradually and in total will be less of a change than was experienced over the past 25 years. Beyond that, the pace will quicken but will be no more dramatic than the 1950 to 1975 period. The total number and proportion of the aged in society will be that much larger and we cannot discount the impact of this change. However, accommodation to this increase should be manageable within what can be regarded as normal changes within a market to altered need and demand patterns. These population changes will occur at a reasonable rate, and adequate lead time is available without a need for crash building or training programs. With or without expected changes in the delivery system, planning and providing for the health care needs of the elderly should not be a major problem because of the anticipated pace of the population age changes. However, the direction and degree of change in general characteristics of the delivery system will be the most significant factor affecting the patterns of availability and use of health-related services and facilities for the aged.

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Discussion

Waldron: You were advocating more widespread use of HMO's?

Brehm: I am saying what I anticipate will occur in areas that I think are appropriate for research. I think we should have more use of HMO's.

Waldron: Is there any evidence at this point that, in such places as the Kaiser plan where you have lower hospitalization rates, there has been a positive effect on any concrete health measures?

Brehm: To my knowledge, there are no data available that indicate either a positive or negative impact of the extremely low rates of inpatient care use. The implication comes through then that if ambulatory care use is a whole lot cheaper, why not use ambulatory instead of inpatient care?

Speaker: Can I ask, in that connection, whether any HMO plan has a lower premium than a fee-for-service plan?

Brehm: There are as many different organizational and financial mechanisms among HMO's as there are HMO's in the United States.

Speaker: Do any of them come out lower than a fee-for-service plan?

Ostfeld: The Harvard Community Health Plan. One thing you must also take into consideration is what are you getting for your money, how much are you paying in the way of premiums, how much service are you getting, and how much out of pocket costs are there? the cost of the premium is just one part of the whole issue.

Kovar: We are talking about health status in using an HMO. Primarily the HMO experience to date has been on younger populations and very few people 65 and over are included in HMO's. This came as a result of HIP in New York and Kaiser on the West Coast which were designed primarily for working populations. Therefore, they were highly selective for young adults who were capable of being employed. It would be very difficult to use those data to evaluate whether the health status of the entire population—and especially the elderly—is affected by belonging to an HMO rather than a fee-for-service plan.

Brehm: Let me respond to the coupling there, which is not an unusual coupling, of HIP and Kaiser. They are about as far apart as organizations go, in financing mechanisms, etc. Therefore there are as many differences in kinds of utilization rates between Kaiser and HIP as there are between HIP and fee-for-service programs. HIP does not employ its own physicians, it uses a panel, and does not own its own facility.

Weksler: I am concerned about the advocacy for increased care for the older populations where presumably the reasons (the health reasons are less fixed) have not been really convincingly shown to attack and maintain health. The question that comes up is in more favorable populations, if health maintenance cannot be demonstrated to be the product of these HMO's, is there really a reasonable and rational belief that HMO's will be more effective in older patients in whom impairments are probably more fixed.

The second point is that the HMO's are presumably evaluated not only on their capability for health maintenance, but ultimately that they will become cost effective. I think that is a question that has to be addressed—whether the cost effectiveness of HMO's for older age groups is a conceivable outcome. I think there are real questions about extending care to older people as opposed to studying diseases and problems of old people. Certainly maintaining health of older people is very expensive technology. I think what I would like to see is that we learn more about gerontology so that we might give preventive health care—I am not sure that we should go the HMO route—to those age 65 and above. I feel that way because there is so little evidence that HMO's are either cost effective or health effective.

Brehm: HMO's have two separable aspects. One is an orientation to preventive care as a presumably more effective technique than getting in after the fact and having to do something about it. There is a whole aspect of HMO's which simply says if you provide a reinforcement such as a reward for efficiency of operation for cost savings, that by giving the physician an opportunity to share in the cost savings by providing prepaid capitation arrangements, you have some built-in bases for reducing the use of the most expensive facility you think you need. That, I think, is a separate thought from whether preventive health care makes any sense or not. There are data on the Kaiser experiments with multiphasic health screening which indicate that to some extent it has a beneficial effect through early detection and use.

Kovar: Do you have any idea what proportion of the health care of the elderly is devoted to prevention as opposed to improving conditions or alleviating symptons or curing?

Butler: It seems to me that Dr. Weksler's question really points to the need for research into the preventable causes for these conditions.

Brehm: I am advocating HMO's as an organizational mechanism which has a built-in base for providing some cost efficiency as opposed to a fee-for-service arrangement whereby the more services the patient uses, the more the physician receives. The way most physicians in the United

States are currently paid, they get nothing if they provide no services. Whereas physicians on a salary base at an HMO are paid anyway. If you do not provide services, you have more money to split up at the end of the year.

Weksler: I really would just like to say that I think the battleground for the HMO should be in the younger population.

Brehm: You and I disagree only in the sense that you are interpreting my comments to mean that the preventive health approach is very meaningful for an already aged population. That is not the basis on which I am advocating HMO's. I am advocating HMO's as a way of avoiding fee-for-service which provides no incentive for efficiency, and instead provides a prepaid arrangement whereby the fewer services a salaried physician can use, the more money stays with the HMO. The experience with those HMO's that own and operate their own facilities and pay physicians on a salary basis with a profit-sharing arrangement is that they seem to use less of the most expensive facilities. Whether the preventive health approach is going to work or not, I will defer to further research as a very specific medical question.

Weksler: Of course, one problem with HMO's is that there is certainly incentive for not using expensive forms of medical, diagnostic, or therapeutic intervention. I agree with you that it is not clear it is beneficial to the patient. It is beneficial, however, for the end of the year statement for each physician.

Brehm: There are lots of differently organized HMO's, some of which have very good organizational bases. While there is no indication that the patients come out much better, there is no indication that they come out any worse.

Closing Remarks by Chairperson Don C. Gibson, D.V.M.

The presentations and discussions in this segment of the conference suggest that application of methods of epidemiology enhances nearly every aspect of research on "Health Care Implications for the Aged." The nature of the data and projected trends presented by conference participants indicate substantive changes over the next several decades in growth in numbers and in relative proportion to the rest of the population of the aged and in the economic, environmental, social, and health care needs of old people in this country.

It appears that the overall life-style patterns, along with utilization patterns for public and private health care programs, will change for our Nation's elderly. All of these variables will have major health care implications for the future.

Conference participants spoke to three broad areas of epidemiologic application:

(1) Better definition of the magnitude of the problems we are about to face as a result of the rapidly changing demographic picture for the

aging population in this country, including anticipated population shifts we will experience over the next several decades.

(2) Definition of problems and approaches we may take to acquire additional information and hence provide much needed types of health care. Here, we must also consider and resolve the complex interrelationships between the need for and the utilization of health care.

(3) The indication that organization and financing of health care for the aged implies a research concept that is multidimensional in character and deals, predictably, with the complex patterns of population differentials in health care use and need for services and facilities for the aged.

Participants also considered how national health care programs impinge on delivery and use of health related services by this segment of the population.

Session V

Summary

Five-Year Perspective on the Elkridge Conference

Adrian Ostfeld, M.D.

Yale University
School of Medicine
60 College Street
New Haven, CT 06510

My remarks are divided into three parts. First, what were some of the more obvious outcomes of the first conference held on the Epidemiology of Aging nearly 5 years ago? The principle aim of that conference was a very simple one; to try to increase the number of epidemiologists and through them their students and colleagues who were concerned about methodological and substantive issues in research on aging in man. The extent to which that goal has been met is substantial. There have been two editions of the book* coming from that conference. There have been creditable applications to the National Institutes of Health. I think it is fair to say that the demographic analysis prepared by Jacob Siegel from the Census Bureau data was to some extent stimulated by the conference. In addition, there was increased attention to updating this information as a basis for planning for the needs of an aging population.

Two conferences in addition to the present one were direct consequences of that first one—a Conference on Senile Dementia and another on Food, Nutrition and Aging. It is too early to tell what the outcomes of these conferences will be. I was not present at the Conference on Senile Dementia and I speak only from second hand sources of information.

The Conference on Food, Nutrition and Aging concluded generally that the disorders of overnutrition were more significant than the disorders of undernutrition among the problems of the aged. A useful publication has resulted from that conference (1).

The 1972 conference led to an increased emphasis on the study of a single phenomenon in stroke which had, what one might call, a negative result; but nevertheless a very useful one. Some unknown proportion of strokes in older persons are reported preceded by so-called early warning signs or transient ischemic attacks and it was a matter of considerable importance to determine the extent to which that was true. In other words, it was important to know what proportion of

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*Ostfeld, A. M., and Gibson, D. C., Eds.: *Epidemiology of Aging* U.S. DHEW, PHS, NIH, NICHD, 1972. Bethesda, MD 20205.

strokes were preceded by signs or symptoms that would "tip off" both the patient and the physician allowing for the possibility of doing something before the stroke occurred.

The data collected, in part as a result of that conference, made it clear that the annual incidence of these transient ischemic attacks or early warning signs of stroke is of the order of 1 per 1,000 per year in persons aged 65 to 74 and in comparable populations the incidence of stroke is 1 to 2 per 100 per—year or at least 10 times as high. Thus, it is clear that the overwhelming majority of strokes are not preceded by these attacks or it may be that our methods of determining their incidence or prevalence require further precision. I am inclined to think that it is the former. At least we know that at the present time there seems no justification for major attempts to educate either the general public or the physicians about early warning signs of stroke; a useful, although essentially negative finding.

As a result in part of the first conference, the Gerontology Research Center is planning to expand its efforts in human aging. Interestingly enough, Dr. Gibson and I have had a total of 35 communications from institutions indicating either that they intend to use the contents of the book in a course or asking permission to do so. (There are still pleasant and naive people who write and ask for permission to do that.) Obviously many other people may have decided to use it and did not notify us of their wishes. These 35 letters have come from baccalaureate degree programs in colleges and universities, from schools of public health, from graduate programs in biology, psychology, one high school, one dental school, but only one medical school.

The second general area that I want to talk about deals with the 14 research questions that arose at the end of the 1972 conference. We might go through them briefly to see the extent to which either new data bearing on these issues have been produced or a reorganization or rethinking of the available data have been accomplished in the intervening years.

1. **Question:** Is there a valid and reliable definition of aging independent of the common chronic diseases?
   Answer: No.
2. **Question:** What does the remarkably high prevalence of carbohydrate intolerance in the elderly mean in terms of morbidity and mortality? What is its mechanism and should it be treated?
   Answer: There has been a modest increase in knowledge in that area. It is clear that the prevalence is indeed high. There is also some evidence that if it is not producing symptoms and if it has not been diagnosed clinically this so-called chemical or clinically inapparent diabetes or abnormal glucose tolerance appears not to have the kind of grave implications for morbidity and mortality associated with clinically evident and treated diabetes. Therefore, there may not be quite the urgency for concern about the medical management of the disorder.
3. **Question:** What are the precursors of Alzheimer's disease and chronic brain syndrome in the elderly?

Answer: Unfortunately, we have made no substantial progress in that area in the last 5 years.

4. **Question:** In elderly cohorts, how do endocrine and immune functions decline over time, and how is the decline correlated with morbidity and mortality?

   Answer: I think a good deal of interesting and intriguing cross-sectional data have appeared about immune function in man. Unfortunately, we have not had quite the same kind of increment in knowledge about endocrine function in man, but it is very difficult and probably unwise to extrapolate widely from cross-sectional studies. I have to say that the information we have is still quite meager and requires replication in longitudinal efforts.

5. **Question:** What is the value of intervention studies on blood pressure in older populations in reducing the incidence of stroke and senile dementia?

   Answer: I think we have had some clear additions in our knowledge in this area. As a matter of fact at the time that the conference was going on, the celebrated VA studies on the treatment of high blood pressure had been published in part and some additional information was going to be published. I think some of the data coming from those studies have had very clear implications for the treatment of high blood pressure in elderly people.

   Two VA studies together treated patients with diastolic blood pressures ranging from 105 to 129. In separate reports from the the VA, there were approximately 80 hypertensive persons in the age group 60 and over studies, with the results of the treatments for hypertension in them remarkably clear. Approximately 40 persons who were treated with placebo in the period of the study, showed 19 major morbid events—stroke, coronary heart disease, congestive heart failure, and three deaths. The group treated with an active preparation, showed no major morbid events and one death in the period of the study. I think this constitutes a substantial increase in our knowledge.

   We have very strong evidence then, that there is value to intervention on blood pressure in older populations. Whether or not the subsequent lowering of blood pressure will retard the decline of the intellectual function that characterizes senile dementia is a question that will require further work and should have a high priority.

6. **Question:** Why is lifespan so extraordinarily short after the onset of senile dementia?

   Answer: Some of the data that Dr. Gruenberg showed us suggest that perhaps the lifespan for those people is beginning to increase somewhat. It still remains very short, however, and the reasons for this abnormal shortening in lifespan are totally unclear. It may be related to such simple measures as nutrition, voiding and hygiene or it may be related to much more complex influences of central nervous system and brain function on a wide variety of bodily systems. I think that we could learn a good deal more in this area by the simplest of bedside observations.

7. **Question:** What living arrangements for the elderly will produce the lowest morbidity and mortality and the highest indices of life satisfaction?
   Answer: I think we phrased this question too simply; we were naive. Those of us who were involved in that conference have learned what many of you must have known previously, that the living arrangements for the elderly having beneficial effects vary with the social situation, the characteristics of the elderly, their past life history, their past experiences and their perceptions. Nevertheless, it should be possible to define these kinds of circumstances for a number of individual elderly groups. I think, therefore, that the question raised less naively has substantial merit.
8. **Question:** What aspects of socioeconomic status make it so important a determinant of longevity?
   Answer: I am afraid we do not know the answer to this question. It is a very difficult question to operationalize. We all recognize the high correlation between variables of socioeconomic status and health and longevity and it is very difficult to understand why this relationship exists. I would like to see a good deal of thought and attention given to operationalizing these questions, defining the variables of moment and consideration given to their being either added to ongoing studies or applied in several settings. Obviously, this is not the time—at the end of the conference and in our present state of fatigue—to spend a great deal of time trying to make these issues more specific. That will have to be done in our own heads when we get back into our own workshops.
9. **Question:** What can we learn from studying the effects of retirement, relocation, bereavement, and economic loss in cohorts of elderly persons?
   Answer: I think today's conference has indicated that we have learned a modest amount in these areas in the time between the conferences. This has been a useful field for investigation and I think that it represents one of the areas in which theoretical and practical progress has been made.
10. **Question:** Why are mortality rates lower in the Mid-western United States than in the Southeast and Northeast?
    Answer: There are many ideas about why this is so, ranging from differences in racial and ethnic composition and in life-style to differences in medical care. However, I believe that at the present time we are limited to speculation. If these differences become smaller, then perhaps the question will become less important. At best, the differences are marginal in terms of their importance because there is not a huge difference between the best and worst State in the United States. If they persist however, or if they increase, I believe they deserve our serious attention.

11. **Question**: What are the differential effects on aging of sex?
Answer: I think we have had increasing information in these areas, even in the last 5 years. Many of these effects have been discussed today in the form of the nature-nurture problem. The consensus now is that the survival and longevity of women appears to be as much or more related to biological factors than to socioeconomic and psychological factors, although the latter unquestionably play some role.

How to proceed beyond this or to define the specific aspects of women's biologicol superiority is a very difficult question. Some of the data presented by Kannel suggested that whatever this biological superiority is, it is present during the reproductive years and dissipates rapidly thereafter. This only helps us to understand the period of life during which it may be operating but it does not necessarily tell us what it is. I believe much thought is necessary before one can operationalize an experiment or a series of observations which will tell us much more about this phenomenon.

12. **Question**: Do dietary habits adversely or favorably affect morbidity and mortality experience in populations?

Answer: Surprisingly, we cannot give a direct answer to this question in spite of the great deal of attention given to feeding programs for the elderly. I still believe that the effects of overnutrition in the elderly are much more significant than those of undernutrition and the 10-State study of relatively impoverished persons, particularly the elderly, which was discussed at the 1972 conference, suggests that the major disorders of malnutrition are neither highly important nor widely prevalent problems in the elderly. They are moderate problems, not grave ones, suggesting to me that the major benefit of feeding programs for the elderly may well be social rather than nutritional.

13. **Question**: Can Comfort's measures provide a useful index of aging?
Answer: No.

14. **Question**: Can studies of aging be grafted on to existing cohorts; for instance, the Framingham, Albany, Evans County, and Tecumseh studies?
Answer: They have been and I am sure that at the time of the first 1972 conference there were already not only substantial plans to do this, but some grafting was already being accomplished. The large scale cohort studies to which I refer are now endangered species. I think that all but Framingham and Evans County are either moribund or dead. The answer to the question "can studies of aging be added to cohort studies of aging populations?" is a resounding yes. Not only has this been done but done with great merit. Further continuation of these studies is vital.

I come now to the third and last parts of my comments. If we have another conference in 5 years, I would like to see a number of events take place in the years between now and then.

First, I want to discuss again the matter of grafting studies of aging onto ongoing cohort studies. If we want to understand health and disease in the aging man or woman, we are going to have to support the few cohorts that now exist to obtain information efficiently and cost-effectively and without a long lag period. I think it is extremely important for the Framingham study, for instance, to be supported well into the future. By that I mean plans should be made now for the next 20 years at the very least. It is a national resource and I think it is extremely important to insure its continued health and survival. Within the limits of both policy and fiscal restraints at the Federal level, security for the investigators and stability of funding should be guaranteed. If this is not done, we will miss our best chance of obtaining cost-effective information relating to man.

I still believe that if it were possible to carry out a cohort study of a population largely free of atherosclerosis, it would be a very worthwhile investment. If such a group can be found and if difficulties encountered can be surmounted, it would be a very rewarding effort in aging. I do not mean to downplay or denigrate cross-sectional studies; they have merit and are easier, shorter and less expensive to do. In my enthusiasm for maintaining the cohort studies that we now have, I do not mean to imply that cross-sectional studies have no value.

Second, it seems to me that during the next 5 years or less we should try to abandon attempts to define aging, functional aging or otherwise. Such attempts have not benefited us in the short run and I do not think they will. We must recognize that what is aging to one generation is perceived by the next to be disease. The hardened arteries and slight mental deterioration that were believed characteristic of aging in the 19th century and the early 1900's, we now recognize as disease.

The same sort of thing happens frequently. The neurological literature is replete with the fact that in the neurological examination of elderly people certain changes are observed. The knee and ankle deep tendon reflexes are absent; the perception of position and vibration sense in the ankle is absent; and the lower legs get weak. These signs are all assumed to be a part of normal or natural aging. However, when we looked at these data, cross-sectionally in a cohort of 3,400 people aged 65 to 74, all of these "hallmarks of the aging nervous system" were largely restricted to persons with diabetes, known coronary heart disease, peripheral atherosclerosis and significant high blood pressure. Persons free of these disorders did not have absent knee and ankle jerks or absent position and vibratory sense and weak lower extremities. Thus another so-called characteristic of normal or natural aging is discarded (2).

Third, much can still be learned by studying the natural environment of aging populations. We need a good deal more information about the effects on aging populations of crime, retirement, bereavement, social isolation, migration, and the like. How do these affect human morale, health and survival in the elderly? Opportunities are arising constantly; we must continue to mobilize our resources and take advantage of them.

Fourth, there ought to be application of epidemiological methods to studying a wide variety of social programs now in use for the elderly. How good or what benefit are such programs as, the Retired Senior Volunteer Programs (RSVP), Foster Grandparent Programs, Hot Meal Programs, or Senior Centers? We can use the methods that we know to determine whether these programs are achieving the goals that they should be achieving.

Again and again we hear the comment that depression is a very serious problem in the elderly, although none of us have data on its prevalence. I think at least five people who have been speakers at this conference and others who have been questioners have raised the same point. My own view is that depression in the elderly not only slows the machine, but wrecks the machinery and we have got to learn more about it. I think that we can probably learn faster if we abandon Freudian theory and complex theoretical frameworks and study it in as clearly defined and as simple a frame of reference as possible.

I have only two more points to make in other areas that I think are important. I am becoming increasingly aware that Federal agencies can do things that university people can only do with difficulty if at all. The Census Bureau, the Framingham study, the Social Security Agency and the National Center for Health Statistic's scholars do things well that universities cannot.

The Federal agencies must be increasingly responsible for providing data on national health needs both met and unmet; on the monitoring of effectiveness of major health programs, both preventive and therapeutic; and for developing more information about health planning for the elderly and for all age groups. I cannot see how scholars in universities can do these jobs at the cost and with the effectiveness that the Federal agencies can. Some things may be accomplished by a Federal agency with a moderate change in budget and personnel that cannot possibly be achieved by any university or consortium of universities. Perhaps programs in which people in the Federal agencies could spend a year or two in universities could be expanded. Likewise, the people in university centers might learn a good deal and might make useful contributions if they had wider opportunity to spend modest periods of time within Federal agencies. Those from the university centers would have a good opportunity to learn the methods and the techniques of the agencies, and those from the agencies might be able to gain knowledge in some areas not available in the agencies. I would suggest that a good deal of thought be given to the development of such programs so that these changing relationships might become possible.

Finally, there is a vicious cycle in physicians' attitudes about the management of all disorders of the elderly. In some cases it makes no difference because there is little they can do. In other cases, however, there is a good deal that they can do. The typical physician who gives care to older patients has been known to say, "There is a great deal of illness and misery among the old folks. I guess that is a part of growing old. I don't think there is a lot I can do about it and I probably shouldn't try too hard to treat it. I might do more harm than good. My older patients certainly get sick and die at a rapid rate. I guess I was right, there isn't much you can do about it."

Such statements make us realize how pernicious this sort of thinking is, particularly for those illnesses about which we can do something. For example, I will take one disorder that I know something about and one about which something can be done. I refer to the treatment of high blood pressure for which Kannel presented evidence. This is a very serious problem in older people. Even if the doctor had available to him the information we have about high blood pressure from epidemiological studies, he might still say, "Why half of my patients 65 and over have high blood pressure. I seem to remember that blood pressure or high blood pressure is 100 plus your age, so I guess that is a part of growing old. Maybe they need that increase in order to maintain circulation of the brain. If that is the case I should not try to treat it very heroically, I might do more harm than good." Or he might say, "My older patients are in and out of congestive heart failure every other week and they are dying like flies of stroke and heart attack. Well, I guess I was right, there does not look like you can do much about treating high blood pressure in older people."

If we want to have an effect on what physicians do we will achieve it by raising the expectations of older people. The final suggestion I have is that the Institute on Aging might define its clientele or its political clique or its sources of political support to try to find out more about what its clients want and to see if they can provide more support for the goals of the Institute. I think we must raise the expectations of the elderly as to what can be done for them in certain specific areas. The expectation that nothing can be done is now more harmful than beneficial. At the time when there really was not much that could be done it was at least, if not beneficial, not harmful. In essence, the goals that I am presenting are related to education of the public and to preventive medicine.

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Discussion

Dr. Butler: I would like to ask Dr. Ostfeld to comment on the potentialities of random preventive trials for some of the suggested interventions to improve the health status of the aged as a quick way of advancing knowledge in addition to passive studies. Why do we put off preventive trials to the last stage for such things as social clubs and other benign activities when we do not need to do the passive studies first.

Dr. Ostfeld: I think that they should be done and I think the need is urgent. The model to which I referred, the VA study, just happened to have some older people in it. No deliberate effort was made to seek them out. We do need controlled clinical trials using older patients not only in the traditional hospital-patient-doctor settings, but in large groups of people in naturally living communities, elderly housing projects or retirement centers. In fact, we need two kinds of clinical trials and I agree very strongly with your comment.

Recommendations for Future Research

Manning Feinleib, M.D., Dr. P. H.

National Heart, Lung, and
Blood Institute
National Institutes of Health
Bethesda, MD 20205

In this section of the summary, I would like to discuss some basic concepts which stand out as a result of our deliberations and recommend some priority studies for future research. Dr. Ostfeld has already proposed the vast majority of the latter, but he was kind enough to leave me two or three.

What can we hope to accomplish in the future in the field of aging? To begin with, we must agree on the concept of what constitutes aging—is it a normal, inevitable condition which parallels chronologic age or is it an avoidable pathologic process? If we think of aging in terms of a population survival curve, we might try to describe an "ideal" curve. One such concept of the ideal might be analogous to Dr. Oliver Wendell Holmes' wonderful one horse shay that looked and functioned beautifully until, at the age of 100, it collapsed, every part at the same time.

If we examine the current population survival curve for the United States and most industrialized countries, it would look like curve A in the figure. There is relatively high mortality during the first year of life, relatively low mortality for several decades thereafter, and then gradually increasing mortality with dramatic decreases in the proportion surviving in later years. The "ideal" curve, however, might approximate a square wave where no one dies until some appropriate age X (curve I in figure V–1). What constitutes an appropriate age for death is obviously an exceedingly complex question involving philosophical, religious and social considerations in addition to biomedical factors. As Dr. Siegel mentioned earlier, as we approach this "ideal" will we have to consider some form of suicide education and planning?

We may have already come closer to this "ideal" curve than many people realize. During the 20th century there have been tremendous gains in life expectancy at birth and also in life expectancy among those who have already achieved middle and old age. A glance at a hypothetical survival curve of about 200 years ago and for some nonindustrialized societies now, as approximated by curve B in the figure, may give some evidence of how far we have come to achieving the "ideal."

These achievements are inherent in any concept for improving the longevity of the aged. Such reasoning could also be applied to general morbidity and to mental and functional ability of the aged. In fact, such a demographic concept is basic to any research aimed at solving the problems of the aged or improving their condition.

The second concept I want to stress relates to health care and services for the elderly in relation to cost-benefit evaluations. We must bear in mind that as we keep people alive longer, we will be performing

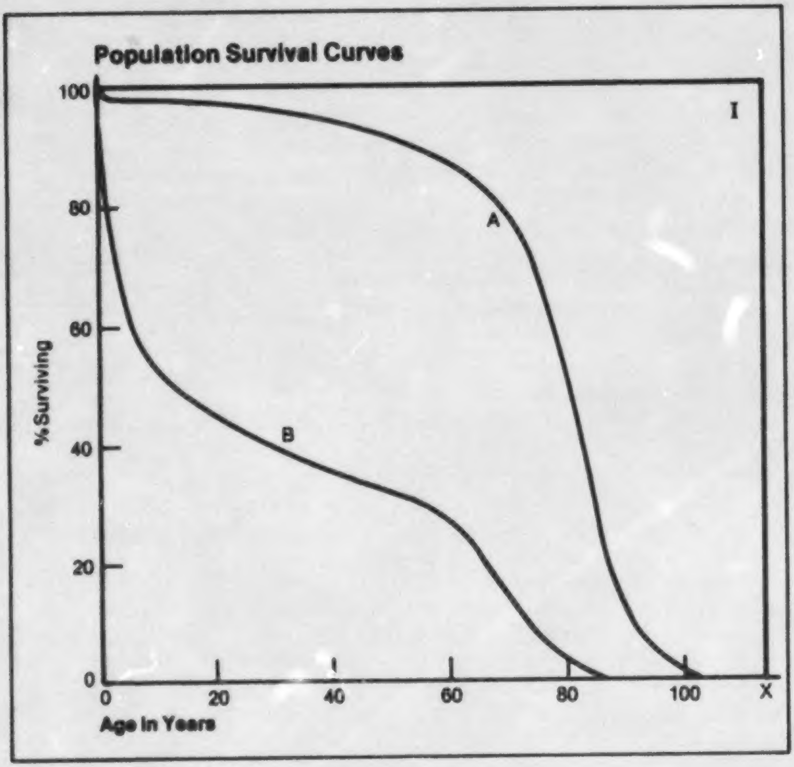

Figure V–1.
Population survival curves.

many more procedures for diseases associated with increasing age—more prostate operations, more cataract operations, more hip replacements, etc. We will be obliged to devote a greater proportion of our resources to restorative procedures for the elderly. Thus, if we eliminate or postpone the major causes of death by means of effective primary prevention (e.g., reduction of risk factors for coronary heart disease and stroke, elimination of environmental carcinogens) or better treatment of early stages of disease, it may not be cost-effective in terms of how it affects the total health care expenditure. In any discussion, therefore, of cost-effectiveness of programs to improve the survival of the elderly, we must be aware of the increased costs necessitated by higher morbidity rates in the elderly and that this will prevail regardless of the methods used to provide health services.

A third concept, which I feel has importance in the light of our discussions here, concerns our need to be aware that the problems we seek to solve are not limited to the aging population The information that has resulted from various research programs involving the general population may be applied immediately to the aged population and may have a relatively high payoff in a very short time. In the management of accidents, for example, our current knowledge of technology applicable for the population as a whole helped solve some very practical problems for avoiding accidents among the aged. However, in the discussion of medication for hypertension among the aged, we find a different problem than that encountered in the younger age groups. In fact, the whole range of therapeutic medications, in their application to the aged as compared to the young, must be studied carefully.

I would like to discuss briefly some items offered by our speakers during the course of the conference which may be listed as suggestions for future studies. I would like to highlight a few of these from the viewpoint of the epidemiologist.

With regard to the biologic correlates of aging, the immunological methods, the enzyme systems, and the like, I think we should give more attention to developing a methodology to enable us to apply these techniques to free-living populations such as those studied in the Framingham project and other large populations. I am aware that some of the data presented included only a dozen or so patients. We must develop the technology necessary to conduct some of these studies using much larger cohorts, possibly in the thousands, to be able to answer the question of the extent to which the biologic factors correlate with other factors of ill health in the aged population.

So far as defining functional ability of the aged is concerned, we recognize that this is both an important social question as well as an important biological one. For example, what should an appropriate dependent variable be in trying to define functional ability. Undoubtedly, different types of definitions are needed for different types of problem-solving conditions. The kind of dependent variable we need for health care systems might be very different from the kind we need to solve the problem of fitness for working or remaining in the labor force. It might be best to use a multivariate approach in both independent and dependent variables to come to some decision on assessing functional ability of the aged.

I would like to emphasize what has been said about the need for studying the natural history of aging in diverse populations such as different ethnic groups, migrant groups, and the like. We should study those that have different rates of longevity, such as Asian-Americans who are the longest lived subgroup in the United States. With regard to migrant groups, we must remember that migration can be viewed from many angles. We discussed the comparison of mortality or longevity in terms of the place where a person was born and the place to which he moved or relocated. We must also be mindful of the situation, not very rare today, where an individual stays where he was born but the community around him changes, i.e., the person does not migrate, the community migrates. A person can find himself in varying socioenvironmental situations without ever changing the place where he lives—the change takes place around him. This seems to me to be an important area of study in terms of the question of relocation or migration and its effect on the aged.

Finally, I would like to repeat some remarks made by Dr. Ostfeld because I think their importance allows for their repetition, especially with regard to the programs of the Institute on Aging. There is a definite need to think in terms of long-term studies. Such studies imply a commitment both for personnel and financial support over many years. If we try to rely on cross-sectional or retrospective studies, we will probably never get the information we really need because predictive factors must be obtained while the individual is healthy and vigorous, and may not be measurable after debility has occurred.

This Institute should assume the responsibility for designing mechanisms, or at least an organizational framework, to allow for scientists to take advantage of any special situations or changing situations occurring in our economic or social setting with a minimum of delay. To continue to undergo the experience, as has been described, of several years of review before we can study, say, a community slated for urban renewal, deprives us of the opportunity to study the problems of relocation. A mechanism must be developed so that we may effectively enter such a community early to take advantage of these situations.

To broaden the sources of information, the Institute should develop a system of coordination with the legislative, regulatory, and service agencies interested in the problems of aging. For example, collaborative studies should be undertaken with NCHS, SSA, VA and other agencies which may be tackling similar problems from different viewpoints and also for very different constituencies in terms of their objectives. This constitutes a potentially beautiful example of intergovernmental cooperation which could work to the benefit of all.

In conclusion, let me thank you again for coming to this meeting on such relatively short notice and doing such an excellent job of highlighting the state of the art in this very complex and diverse field.

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